The American Journal of Surgery

ADVERTISERS INDEX

YEARS of laboratory and clinical study have produced this effective vasoconstrictor which will provide more prompt and prolonged relief from nasal congestion. Preferred by physicians and patients because of absence of local and systemic side effects, PRIVINE* Hydrochloride has a more sustained action, outlasting in most cases the effect of other vasoactive fluids.

Clinical solutions have been prepared in line with modern requirements set up for nasal medication by authoritative investigators. They are strongly buffered to resist alkaline pathological secretion and to preserve a normal, slightly acid reaction of the nasal mucosa. Their isotonicity and the reaction of the solution (pH—6.2) are specifically recommended for the restoration and preservation of the ciliary activity and the normal physiology of mucous membranes. The use of PRIVINE Hydrochloride is not followed by a compensatory swelling of the mucosa.

PRIVINE HYDROCHLORIDE is a most economical and efficient nasal medication. Issued as an 0.1% solution, bottles of 1 oz. with dropper. An 0.05% solution is available especially for children.

CIBA *Pharmaceutical Products, Inc*
SUMMIT, NEW JERSEY

Safe · Convenient

SAFER than non-boilable catgut because heat-sterilized *after* the tubes are sealed . . . tubes may be boiled or autoclaved to assure absolute asepsis of their outer surfaces.

MORE CONVENIENT because adaptable to any operating room technic . . . quickly prepared for use, as required, along with instruments or dressings . . . flexibility readily controllable to any degree.

These advantages, always important, assume special significance today when surgical personnel and facilities are under extraordinary pressure.

D & G Claustro Thermal Boilable Catgut

Claustro-thermal Sterilization is an Exclusive Davis & Geck Process

Field Service on
Your G-E Equipment
Will be Maintained

Today's Best Buy – U. S. War Bonds

TO the thousands of hospitals, clinics, and physicians using G-E x-ray and electromedical apparatus, we address this message of assurance: *G. E.'s local branch offices throughout the United States and Canada, which for years past have responded to your various needs, will continue to provide that expert maintenance service so important in today's handling of the health problem.* ◖ Since you probably are working your equipment harder these days than ever before, it is highly important that it be kept working, and we are not unmindful of the responsibility which we assumed when you bought G-E equipment—to help you maintain it in the best possible operating condition. ◖ We give you this assurance despite the difficulties under which our field representatives are working, due to tire and gas rationing and other war-time restrictions. Because these men are determined to justify your manifested confidence in the G-E organization, you'll find them ready and willing to sacrifice even their own time when you are urgently in need of maintenance service. *They won't let you down!*

GENERAL ELECTRIC X-RAY CORPORATION
2012 JACKSON BLVD.   **CHICAGO, ILL., U. S. A.**

The American Journal of Surgery, Inc.

Reg. U. S. Pat. Off., Nov. 3, 1936

Editorials

The American Journal of Surgery

NEW SERIES VOL. LIX　　　　Contents · January, 1943　　　　NUMBER ONE

General Information on Page 4　　　Advertising Index on 3rd cover

General Information

THE AMERICAN JOURNAL OF SURGERY does not hold itself responsible for any statements made or opinions expressed by any contributor in any article published in its columns.

Articles are accepted for publication with the understanding that they are original contributions never previously published. All manuscripts are subject to editorial modification, and upon acceptance become the property of THE AMERICAN JOURNAL OF SURGERY, INC.

A reasonable number of illustrations are supplied free of cost; special arrangements must be made with the editor and publishers for excess illustrations and elaborate tables.

Reprints are furnished on order. Prices are quoted when articles are in page form.

Material published in THE AMERICAN JOURNAL OF SURGERY is copyrighted and may not be reproduced without permission of the publishers.

Change of address must reach us by the 15th of the month preceding month of issue.

PREPARATION OF MANUSCRIPTS

Text. Manuscripts are to be typewritten, on one side of the paper, with double spacing and good margins. The original should be sent to the editor and a carbon copy retained by the author.

Illustrations. Illustrations must be in the form of glossy prints or drawings in black ink (*never* in blue). On the back of each illustration the figure number, author's name and an indication of the top of the picture should be given. Legends for illustrations are to be typewritten in a single list, with numbers corresponding to those on the photographs and drawings. Please do not attach legends to the pictures themselves.

Bibliographies. Bibliographic references should be at the end of the manuscript, in alphabetical order, and not in footnotes. Each reference should include the following information in the order indicated: Name of author with initials; title of article; name of periodical; volume, page and year. The following may be used as a model:

PHANEUF, LOUIS E. Indications and technique Am. J. Surg., 25: 446, 1937.

The author should always place his full address cn his manuscript.

Address all correspondence to

The American Journal of Surgery, Inc.

49 WEST 45TH STREET · NEW YORK

WHEN MORPHINE PROVES A BOOMERANG

Morphine frequently boomerangs—producing undesirable by-effects that often becloud the clinical picture. Pantopon, the Roche sedative-analgesic, is less likely to cause unwelcome side reactions. It contains *all* the alkaloids of opium in a highly purified form, free from irritant gums and resins, affording a smooth pharmacologically balanced action. Try Pantopon in place of morphine—⅓ gr. where ¼ gr. of morphine is indicated . . . HOFFMANN-LA ROCHE, INC., NUTLEY, N.J. *Makers of Medicines of Rare Quality*

TRY PANTOPON 'ROCHE'

Cornerstones

ONLY THROUGH ABILITY TO ESTABLISH AND MAIN-

TAIN HIGH STANDARDS AND TO CONTRIBUTE NEW

AND USEFUL PRODUCTS FOR THE CONTROL OF

DISEASE CAN A PHARMACEUTICAL MANUFACTURER

BECOME A HELPFUL FACTOR IN WORLD MEDICINE

ELI LILLY AND COMPANY

QUICK EPITHELIAL REGENERATION

IN BURNS
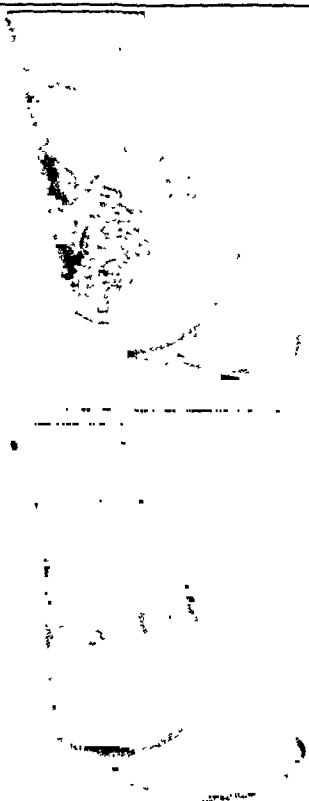
Third degree thermal burn of heel. Treatment with infrequent dressings with White's Vitamin A and D Ointment initiated February 20. No other local treatment was used.

Complete healing as observed May 8. There was no intercurrent local infection. No skin-grafting was required.

An important objective in the local management of burns is the restoration of epithelial tissue in the shortest time possible—without contracture or undue scarring.

White's VITAMIN A & D OINTMENT

helps reduce the period of recovery in burns and other slow-healing lesions by encouraging rapid, healthy granulation and epithelial repair.

White's Vitamin A and D Ointment supplies the vitamins A and D derived from fish liver oils, in a suitable excipient. It is a pharmaceutically elegant compound, possessing a lasting, pleasing odor—remains stable and does not develop rancidity.

Supplied in four convenient sizes: 1.5 oz. tubes, 8 oz. and 16 oz. jars, 5 lb. containers.

Our Medical Division will be glad to receive your inquiry concerning any phase of the use of this preparation, or any other of the extensive line of White's prescription vitamin products. Write White Laboratories, Inc., 113 N. 13th Street, Newark, N. J.

Editorials

THE PROBLEM OF PERITONEAL DRAINAGE

HILTON said, "It would be well, I think, if the surgeon would fix upon his memory, as the first professional thought which should accompany him in the course of his daily occupation, this physiological truth—that Nature has a constant tendency to repair the injuries to which her structures may have been subjected, whether those injuries be the result of fatigue or exhaustion, of inflammation or accident. Also that *this reparative power becomes at once most conspicuous when the disturbing cause has been removed;* thus presenting to the consideration of the physician and surgeon a constantly recurring and sound principle for his guidance in his professional practice."

The above paragraph was not written from the point of view of adequate therapy for peritonitis, because abdominal surgery scarcely existed in the days of Hilton, but the principle expressed may well be applied to intra-abdominal infectious lesions. It suggests that once the disturbing cause is removed Nature may well do the rest. Thus the removal of the diseased appendix, gallbladder, or devitalized bowel in strangulation might give sufficient relief for Nature, unaided and unimpeded, to accomplish the remainder of the recovery. The use of drains began long before the surgeon knew of the existence of bacteria and was emboldened by his knowledge of bacteria and aseptic methods to open the abdomen at will. The use of drains came from empiri-

cism not science and yet was carried over bodily into the scientific era.

Now the question of drainage in peritonitis has become a major problem for the surgeon. Indeed, it is perhaps more of a controversial problem than ever before, and yet this matter of drainage is as old as surgery and has been discussed by our leaders from the very beginnings of surgical practice. Lord Lister carefully discussed the advantages of the primary closure of compound fractures. He based his reasoning for omitting drains on carefully conducted pathological experiments. The matter of nondrainage of infections in the peritoneal cavity was reintroduced as long ago as 1924 by Sir Henry Gray who, fresh from his experience in the Great War of 1914 to 1918, found further support to his prewar experience of omitting drains in cases of peritonitis following acute appendicitis. He initimated, as did Hilton, that surgeons rarely give enough credit to a patient's natural recuperative powers. He described in detail his own technic for dealing with intraperitoneal infection and abscesses, showing that patients suffering from this malady did better if the drains were omitted. He extended his observations concerning nondrainage in appendicitis to peritonitis from other causes such as perforated ulcer or primary pneumococcal peritonitis. In further support of this point of view is the fact that in the days when

typhoid fever was rampant, ruptures of the ileum were closed without drainage.

Now we are face to face with this issue of the use of drains in the peritoneal cavity. The advocates of drainage and nondrainage bitterly oppose each other, basing their conclusions more on limited personal experience than on a scientific study of the immunological and pathological reactions which occur. Thus a surgeon who drains patients with appendicitis cites how all but 1 or 2 per cent recover and does not take into consideration the fact that perhaps a great number would have recovered if he had not left the drains in place. No experienced surgeon should lay down fixed rules for the care of sick people in the hands of another surgeon, but we have a right to suggest that the removal of the cause of intraperitoneal infection is the chief element in recovery and that once this cause has been removed and the resistance of the body augmented by the use of the new sulfa drugs, drains may be considered, as a rule, both dangerous and inadvisable.

ELLIOTT C. CUTLER, M.D.

SOCIALIZED MEDICINE

AN American medical revolution is on its way declared Michael Evans in the November issue of Coronet magazine. The armed forces are taking approximately one-third of our practicing physicians with the result that some form of socialized medicine is coming into widespread being.

By January 1, 1943, almost every doctor under forty-five and many in the forty-five to fifty-five age group, will be in uniform. The situation for civilians, according to Assistant Surgeon General Mountain, may truly be called desperate.

"What's going to happen to us when we're sick?" millions of Americans are asking.

With 50,000 out of our 181,530 registered physicians in khaki and with the concentration of workers in war-industrial communities, problems are arising which must be met, the Coronet article states emphatically.

The answer may be socialized medicine, which the American Medical Association has been fighting so bitterly for so long. Today, however, the A.M.A. has no choice in the matter. Medical care must be reallocated to care for the needs of civilians. The United States Public Health Service and other federal agencies under Paul V. McNutt, chairman of the Manpower Board, are assigning doctors to towns as they are needed, and the American Medical Association is assisting the government, whether it will or no. Recruiting of physicians for the armed services has been placed in the hands of the A.M.A. and under an arbitrary but technically voluntary basis, it is assigning physicians to specific localities. Federal and federally-sponsored clinics may come too.

The American Medical Association is also calling upon people to take care of themselves, to learn first aid, to see doctors either at a clinic, a hospital or at their offices, if they must. In one West Coast city, the A.M.A. is buying radio time to tell the public not to call a doctor unless the need is absolutely imperative.

But these measures, although helpful to some extent make but a little dent in the problem. You cannot drain doctors out of the population at the present rate and expect to escape the consequences.

Socialized medicine isn't here—yet, says Michael Evans; but what is being done by the government with the cooperation of the A.M.A. is closer to it than anything the United States has seen before, and it is obvious that revolution is taking place in medicine today. Whether or not the change will leave its mark on the medicine of the future is still a question, but it is one that is being answered—now!

ABDOMINAL CARDIECTOMY OR SUBTOTAL GASTRIC RESECTION FOR CANCER OF THE PROXIMAL HALF OF THE STOMACH*

CASE REPORT

GEORGE T. PACK, M.D. AND CHARLES S. CAMERON, M.D.

Attending Surgeon, Memorial Hospital for Cancer and Allied Diseases

Fellow in Cancer Research, Memorial Hospital for Cancer and Allied Diseases

NEW YORK, NEW YORK

IN years past, one of the necessary criteria for resectability of a gastric cancer was subtotal involvement of the stomach with freedom of the cardia. This dictum, stated by William Welch in 1885, four years after Billroth had performed the first successful partial gastrectomy for cancer, remained practically inviolate through two successive generations of surgeons. Sporadic attempts at surgical removal of the proximal half of the stomach were initiated by Biondi, who in 1896 first attempted the transthoracic approach. Through a thoracotomy, he freed the terminal esophagus at the hiatus and after delivery of the stomach into the thorax, effected a resection of the cancerous segment and an intrathoracic esophagogastric anastomosis. Chiefly during the last decade, intrepid European and American surgeons have boldly attacked the problem of cancer of the gastric cardia, and by mastering the many difficulties in surgical technic inherent in this location, have brought these 8 to 10 per cent of gastric cancers into the scope of possible cure.

The twenty-five gastric cancers involving the cardia, which have been surgically removed on the gastric service of the Memorial Hospital during the past three or more years, have been excised by five different technics:

1. *Total Gastrectomy—Sixteen Cases.* Even though the involvement of the stomach was subtotal, the extension of the cancer into the cardia and fundus necessitated a removal of the entire organ. Re-establishment of gastrointestinal continuity was accomplished in fifteen instances by esophagojejunostomies and in one unusual case by an esophagoduodenostomy.

2. *Transthoracic, Transdiaphragmatic Resection of the Terminal Esophagus and Gastric Cardia with Intrathoracic Esophagogastric Anastomoses—Six Cases.* This is the classical procedure described and performed by Sauerbruch, Hedblom, Phemister, Ohsawa, Fischer, Carter and others, in which the intact distal half of the stomach is converted into a tube for direct anastomosis with the esophageal stump. The diaphragm is repaired and sutured around the thoracic stomach.

3. *Transthoracic, Transdiaphragmatic Resection of the Terminal Esophagus and Major Portion of the Stomach with Intrathoracic Esophagojejunal Anastomosis—One Case.* This operation, performed by Dr. Gordon McNeer was necessary because of subtotal involvement of the stomach by the cancer.

4. *Transthoracic, Transdiaphragmatic Subtotal Resection of Stomach and Esophagus with a Prethoracic Subcutaneous Esoph-*

* From the Gastric Service of the Memorial Hospital for Cancer and Allied Diseases, New York City.

agoplasty—One Case. So much of the stomach and esophagus was removed that it was impossible to effect an anastomosis

monly done but there are limitations to this maneuver, because of its precarious blood supply and fragility and because of upward
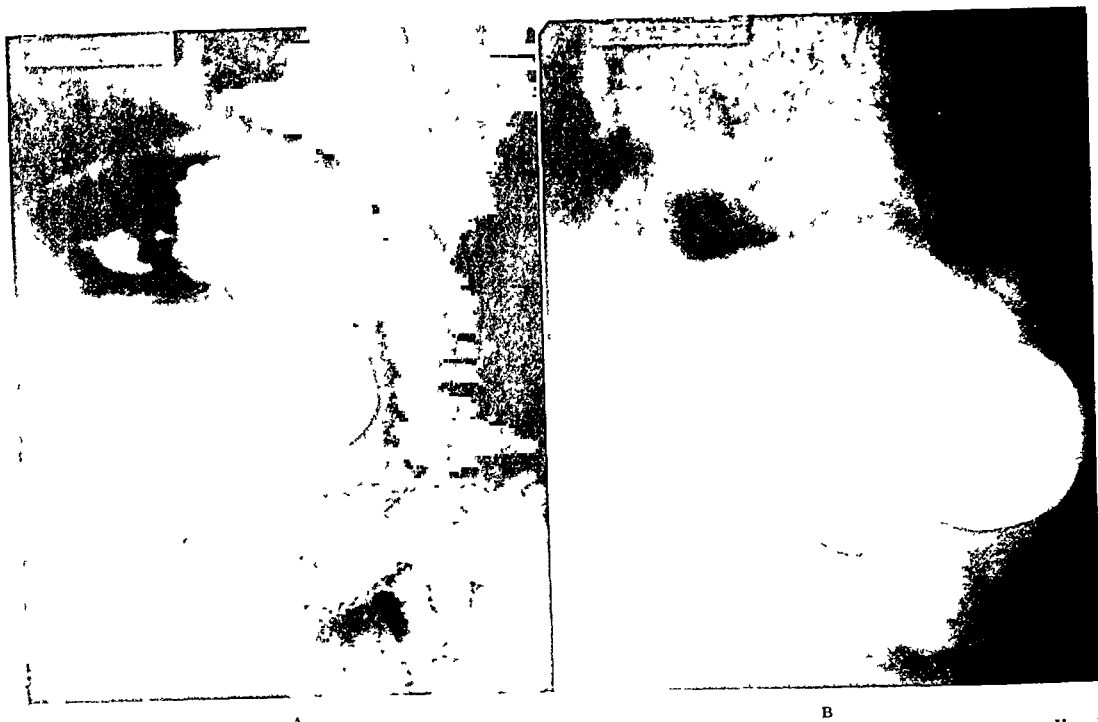
FIG. 1. A, roentgenogram of stomach made prior to operation. The tumor in the cardia may be seen outlined against the gas bubble. B, postoperative roentgenogram. The irregularity along the lesser curvature is the immediate result of suturing with dePetz metal clips. The gastric tube connecting esophagus and pars media has been constructed from the greater curvature of the fundus.

within the chest. By multiple-staged operative procedures, the gastrostomy (Janeway) and a prethoracic esophagostomy were united by constructing a subcutaneous dermal tube down the left wall of the chest.

5. *Abdominal Cardiectomy or Subtotal Gastric Resection for Cancer of the Proximal Half of the Stomach with Intra-abdominal Esophagogastric Anastomosis—One Case.* This case is herein reported.

It is highly desirable, whenever possible to have the line of anastomosis of esophagus and stomach, or esophagus and jejunum as the case may be, below the diaphragm. The repeated successful performance of this anastomosis within the chest by other surgeons and by us, does not contradict the fact that the abdominal anastomosis is safer. Mobilization of the esophagus by freeing it from the diaphragm in order to increase its abdominal extension is com-

invasion by the cancer. Heuer, Andrus and Bell,* being cognizant of these dangers, originated an ingenious two-stage operation in which the transposition of the lower end of the esophagus into the abdomen through the transplantation upward of the left half of the diaphragm, was followed later by the resection of the cancerous segment of esophagus and cardia and an intra-abdominal esophagogastrostomy.

The questions may readily be asked: Why not do a total gastrectomy instead of a subtotal resection of the proximal half of the stomach? Does the preservation of the distal and apparently normal half of the stomach add greatly to the risk of local recurrence? In answer to these queries, one

* Heuer, G. J., Andrus, W. deW. and Bell, H. G. The experimental transplantation of the diaphragm as an adjunct in the treatment of lesions at the lower end of the esophagus. *Ann. Surg.*, pp. 273–286, January, 1925

may remark that surgeons are content to perform partial gastrectomies for cancers in the distal end of the stomach if they are

FIG. 2. Gastric cancer situated in region of cardia. Transsection of stomach between two rows of dePetz metal clips, placed distal to the involved segment.

assured by clinical inspection that the upper gastric segment is apparently normal. The partial removal of the upper half of the stomach in the transthoracic, transdiaphragmatic operations is considered the procedure of choice, yet by the abdominal route surgeons believe they are compelled to sacrifice the entire stomach. Our experience with a single case does not warrant the formulation of any rules in this matter, other than to state that the feasibility of the operation of abdominal cardiectomy should be considered. In our three hundred pound patient, a total gastrectomy would have been fatal because his abnormally short jejunal mesentery offered a major obstacle to the construction of an esophagojejunal anastomosis, even by the Roux en-y procedure. The esophagogastric anastomosis functioned in a manner similar to the normal cardia as did the distal half of the stomach with its intact pylorus. The patients who have had total gastrectomies, invariably have some degree of post-

operative abdominal discomfort; some have strictures and other mechanical disabilities; many have metabolic deficiencies such as
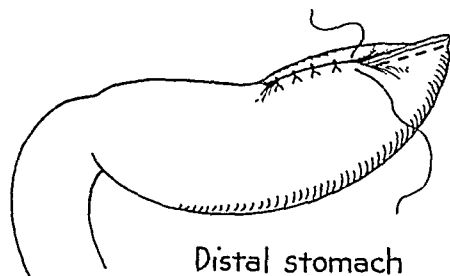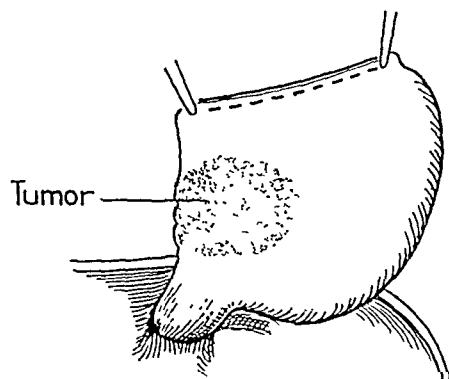
FIG. 3. The proximal gastric segment containing the cancer is elevated to expose the posterior surface of the abdominal esophagus. The distal gastric segment is sutured in preparation of a tube to be used for an esophagogastric anastomosis.

anemia, steatorrhea and inability to gain weight. These attendant sequels should be carefully considered in the elective choice of a total gastrectomy over partial gastrectomy (abdominal cardiectomy) for cancers of the proximal gastric segment.

CASE REPORT

M. Z., a fifty-six year old, white, German, married male applied at the Memorial Hospital on April 28, 1941. His father had died following an accidental injury and his mother had died as a result of "rheumatism." Of five siblings, one brother was alive and well, one brother had died of heart disease, and three sisters had died of causes unknown. The patient was a night watchman. In 1916, cranial decompression was done for a shrapnel injury

to his head. His previous health had been otherwise excellent and he had never suffered from indigestion prior to his present illness.

sclerosis of the retinal vessels. The chest was emphysematous; breath sounds were harsh and râles could be heard throughout the lungs. The
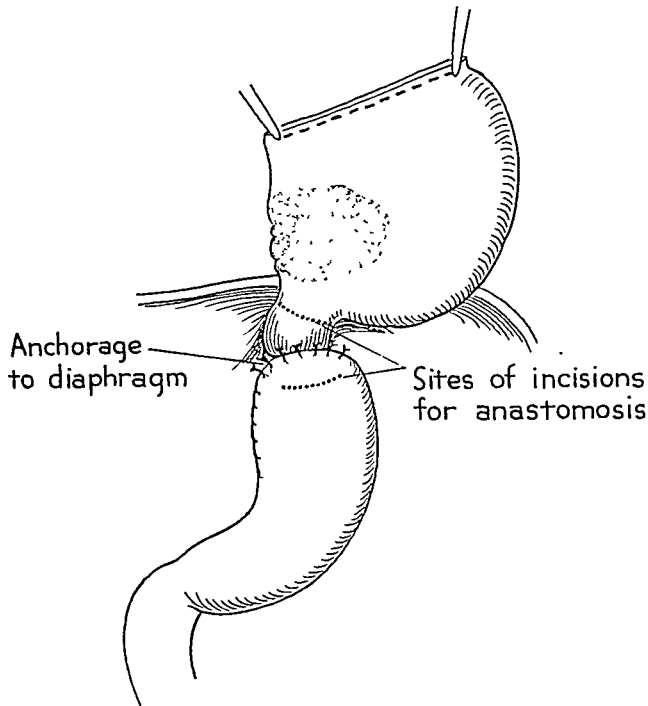
FIG. 4. Diagram showing anchorage of closed gastric tube to the diaphragm and esophagus. Selection of sites of incisions in esophagus and stomach for the anastomosis.

He consumed large quantities of food and drank beer occasionally. His usual weight was 300 pounds.

The onset of symptoms occurred in November, 1940, when he experienced transient cramp-like pains in the upper part of the abdomen immediately after swallowing solid food. This discomfort lasted only a few minutes, presumably until the ingesta had passed into the stomach. During the ensuing five months, the severity of these epigastric pains associated with dysphagia, increased, although he never complained of anorexia, nausea or vomiting. His weight loss of only fifteen pounds was not attended by debility. One week prior to his first hospital visit, he experienced constant pain, mild prostration and the passage of tarry stools continuously for two days. It was this severe hemorrhage which led to his initial gastrointestinal survey.

Physical examination revealed the patient to be an obese white male in late middle life. He weighed 285 pounds. The sclerae were non-icteric; ophthalmoscopy revealed moderate

cardiological consultant observed some myocardial fibrosis and coronary sclerosis. The blood pressure was 170/90 mm. of mercury. The abdomen was pendulous but uniformly soft and without signs of fluid. There were no palpable masses in the abdomen. A bimanual recto-abdominal examination revealed no evidence of metastatic cancer deposit in the rectovesical pouch.

Fluoroscopic examination was unsatisfactory because of the patient's great girth. X-ray films of the stomach showed an unusual configuration of the gas bubble shadow associated with an increased density of the soft tissues medial to the gastric cardia. Slight barium retention appeared in the body of the stomach just below the cardia. The presumptive x-ray diagnosis was carcinoma of the cardiac segment of the stomach.

The interior of the stomach was visualized through a flexible gastroscope. On the lesser curvature between the incisura and the cardiac orifice, a necrotic ulcer was easily seen. The

cardiac orifice and the distal half of the stomach were of normal appearance.

The urine was acid, contained no albumin
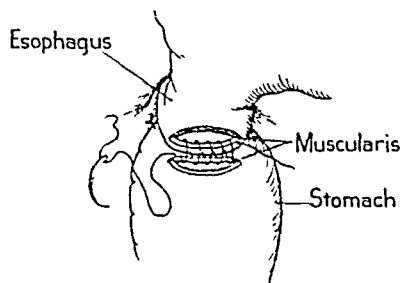
FIG. 5. Diagram to illustrate the technic of anastomosing the esophagus to an incision in the anterior wall of the stomach, i.e., a termino-lateral anastomosis. The incisions are down to but not through the mucosa at this stage.
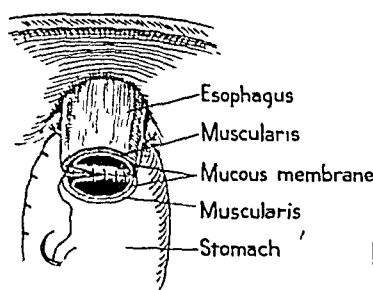
FIG. 6. Completion of posterior layer of anastomosis after deepening incisions through the esophageal and gastric mucosa. The cancer-bearing segment of the stomach has been removed.
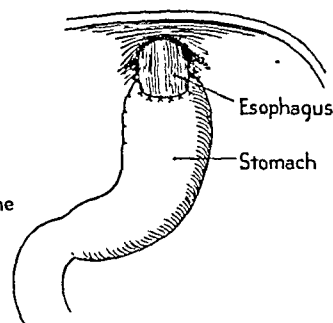
FIG. 7. Diagram illustrating the completed anastomosis.

nor glucose, and showed good concentration by the Fishberg test. The stools were negative for occult blood. Gastric analysis using the ergamine test revealed an increased free and total acidity, the free hydrochloric acid being as high as 121 degrees forty-five minutes after the injection of ergamine acid phosphate. The hemoglobin registered 100 per cent and the erythrocyte count was 4,740,000 per cu. mm. of blood. The white blood count was 5,100 per cu. mm. with a normal differential distribution. The hematocrit reading was 36 per cent. Chemical analyses of the blood were as follows: blood urea nitrogen—18.8 mg. per cent, blood sugar—104 mg. per cent, bilirubin—0.65 mg. per cent, sodium chloride—639 mg. per cent, total cholesterol—194 mg. per cent, free cholesterol—50.5 mg. per cent, cholesterol esters—143.4 mg. per cent, serum protein—7.1 Gm. per 100 cc., prothrombin—90 per cent and blood vitamin A level—110 U. S. P. units.

The diagnosis was carcinoma of stomach, lesser curvature, juxtacardiac. There were no contraindications to laparotomy. The patient was admitted to the Memorial Hospital on June 4, 1941, and prepared for an abdominal operation.

Operation on June 18, 1941, consisted of subtotal gastric resection (cardia) and esophagogastric anastomosis (abdominal cardiectomy). Under spinal nupercaine anesthesia, supplemented by intravenous pentothal sodium, an upper left midrectus incision was made. The liver was found to be of normal size and

consistency without evidence of metastatic cancer. The primary gastric tumor was located in the extreme cardiac segment, closely encroaching upon the terminal esophagus. The tumor was only about 5 cm. in diameter. The involved portion of the stomach could be mobilized readily and the cancer was not adherent posteriorly. After removing the larger part of the great omentum, the gastrohepatic, gastrocolic, gastrosplenic and gastrophrenic ligaments were severed between clamps and ligatures to free the proximal half of the stomach. The coronary and left gastro-epiploic vessels were doubly ligated and severed. A dePetz sewing clamp was now applied across the stomach, approximately in the transverse direction, so that the line of division extended from a point at the junction of the lower and middle thirds of the lesser curvature to a point at the junction of the upper and middle thirds of the greater curvature as indicated in the accompanying diagram. The stomach was then trans-sected between the brads and the distal suture line completely inverted by seromuscular silk sutures.

The mobilization of the proximal or tumor-bearing segment of the stomach was completed with some difficulty. Upward traction of this segment toward the left costal margin in order to expose the posterior wall of the abdominal esophagus after the method of Moynihan was then accomplished. The distal cylindrical gastric tube was then brought up posteriorly and anchored to the diaphragm in order to lessen subsequent tension on the anastomosis, which was to be effected in an end-to-side fashion between the terminal esophagus and

the anterior wall of the residual stomach. (Fig. 4.) A new stoma was made transversely in the anterior wall of the stomach about 3 cm. below the tip of the gastric tube. The anastomosis was completed by the usual technic with three suture lines posteriorly and two anteriorly, the inner one of which was a Connell inverting suture. A Levin tube was inserted through the nose and directed through the anastomosis before the abdomen was closed. The abdominal wound was closed in layers.

A blood transfusion of 600 cu. cm. was given during the operation and 250 cu. cm. of plasma was administered on the third postoperative day. Convalescence was complicated by a mild bronchopneumonia, controlled by sulfanilamide, and by dehiscence of the wound, requiring secondary closure.

Gross and Microscopical Study of the Surgical Specimen. The posterior wall of the stomach contained an ulcer measuring 5 cm. in diameter, with elevated grayish margin and relatively clean base. The upper margin of the ulcerated tumor involved the cardiac orifice and a portion of the esophagus itself, leaving a stenotic passageway of less than 1 cm. in diameter. The gastric mucosa appeared normal with prominent rugal markings. The adjoined perigastric lymph-nodes were replaced by tumor tissue. On microscopical study, the diagnosis was diffuse adenocarcinoma grade 4, with metastases to multiple (3) juxtacardiac lymph-nodes.

After the patient was ambulatory, he experienced some discomfort from a large incisional hernia. His postoperative weight was 238 pounds and this has remained stationary. He complained of postprandial discomfort in September 1941, and roentgenograms made then showed some irregularity of the reconstructed gastric tube which might be either an operative defect or recurrent carcinoma. Because of the great anaplasia of the tumor, postoperative x-ray therapy was administered from which the patient experienced relief of symptoms. The following factors were employed: 1,000,000 volts, filter of 2 mm. Hg., 8 mm. cu., target-skin distance of 70 cm., 3 ma., fields measuring 12 by 16 cm., employing three skin fields, anterior, lateral and posterior. Three hundred roentgens were given daily, alternating until a total dose of 2700 r × 3 was administered. A recurrence of symptoms occurred in August, 1942, and an additional series of x-ray treatments with two-thirds the original dose have just been given.

FISSURE-IN-ANO

EDWARD T. WHITNEY, M.D.
Surgeon-in-charge, Rectal Clinic, Boston Dispensary
BOSTON, MASSACHUSETTS

A TRUE fissure-in-ano is one of the most painful lesions known and even when it has become chronic or is in a remission, there is an underlying pathological condition which is still producing many local and reflex symptoms such as tenderness, splitting at stool, constipation, nervousness and irritability, tension, lack of energy and concentration and an inability to get the mind off the "rectum." It is much more common a condition than is generally supposed. Every sore anus should be considered as harboring an actual or potential fissure until that possibility has been definitely eliminated. Furthermore, recurrences even after surgical intervention are altogether too frequent and nothing is more discouraging than to have a fissure recur after the patient has submitted to some procedure which was supposed to cure him. As Blaisdell[1] has said, heretofore treatment has not been "along logical and scientific lines."

To begin with the term, fissure-in-ano, should be accurately defined. The little splits in malnourished skin at and just outside of the anal verge are not true fissures-in-ano. Neither are the cracks in the bases of pruritic creases. The true fissure-in-ano is intra-anal. Two distinct varieties may be recognized: First, there is the superficial split, abrasion or ulcer occurring anywhere on the anal wall except at the posterior commissure and usually accompanied by some indefinite discomfort only. Second, there is the deep, round or oval ulcer in the anal wall at the posterior commissure producing severe pain at the time of and following a bowel movement. The latter comprise some 90 to 95 per cent of all fissures-in-ano.

The first, superficial type is accompanied by a sore anus but it is only occasionally accompanied by the severe lancinating pain and spasm associated with the second, deep type. Often, the chief complaint is bleeding with tenderness or soreness being elicited only after questioning. Usually, its etiology is fairly easily determined. There are at least four possibilities: (1) a simple split, laceration or abrasion from a constipated stool or foreign body; (2) the rupture of a small thrombosis low down on the anal wall; (3) a split of the intra-anal skin overlying an area of fixed scar tissue occurring either anteriorly, as the result of parturition, or elsewhere, as the result of chronic, inflammatory "cryptitis" and (4) a ruptured but inadequately draining, infected anal duct or "crypt" much like that to be described later on but not involved in the complications so often existing at the posterior commissure. Treatment of such a fissure depends upon ascertaining the existing, underlying, causative factors and eliminating them.

It is the second type which produces the typical, severe and agonizing pain recurring with each bowel movement and lasting for some time thereafter. This pain may be so acute that bowel movements are withheld for days. The victim becomes driven; he can neither sit still nor stand still. When fear of examination prevents medical assistance, a distinct loss of weight may occur. After a few days or weeks under palliative measures this acute stage may quiet down and become chronic or healing may even take place only to be followed by recurrences. Upon examination, it may be impossible to find anything beyond spasm of the sphincter so marked that introduction of the finger into the anus is prevented. However, in the majority of cases, a little edamatous "sentinel pile" will be seen and if this is retracted the distal tip of the
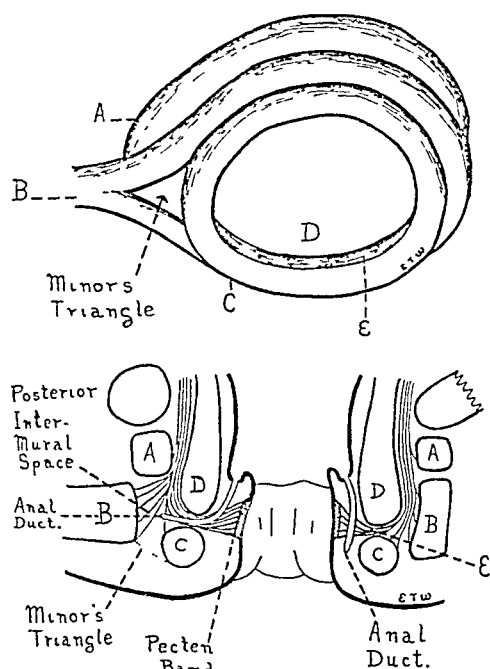
FIG. 1 Diagrams illustrating the various parts of the sphincters. The top one is a modification of the conceptions of Milligan and Morgan and of Blaisdell. The view is as if one were looking into the rectum through the dilated sphincter with the skin and mucous membrane removed. Through the opening is seen the muscular wall of the rectum, the lower end of which is called the internal sphincter (D) while the opening itself is formed by the subcutaneous portion of the external sphincter (C). Between the two is illustrated the intermuscular septum composed of strands of longitudinal muscle (E). The superficial portion of the external sphincter (B) is shown forming Minor's triangle as it goes off at a tangent to become attached to the coccyx. The deep portion of the external sphincter (A) is behind this.

The lower diagram illustrates a sagittal section of the top view with the skin and mucous membrane intact. The lettering is the same. (A) is the deep portion of the external sphincter; (B) is the superficial portion of the external sphincter; (C) is the subcutaneous portion of the external sphincter; (D) is the internal sphincter and (E) is the longitudinal muscle coming down the outside of the rectum with some of it passing through the intermuscular septum to become attached to the anal skin in that location. The left half of the diagram is at the posterior commissure and shows a crypt of Morgagni into which an anal duct empties with its tip lying in the intermuscular septum. The right half depicts the usual location of an anal duct not at the posterior commissure.

fissure may be visualized. After relaxation of the sphincter is obtained and a speculum inserted, the fissure will be found to be round or oval and the base will be seen to be composed of either firm, white, fibromuscular tissue[2] or the external sphincter[1,3] or both. In other words, except while the contracted anus is pinching it together, it is not a fissure at all but rather an ulcer in or through the intra-anal skin.

This second type occurs only at the posterior commissure and this fact calls for careful examination of any especial conditions or pathological condition existing solely in the latter region. There are three such factors which require consideration, two of them are pathological and one is anatomical. The first and most important pathological entity to be found in this region is the presence of or the remains of an infected anal gland or "crypt" (so-called). This, in turn, affects and is affected by a pathological structure commonly known as the Pecten band. However, both of these factors may be found present elsewhere in the anus and only rarely do they produce any such syndrome of signs and symptoms. *It is the peculiar anatomy of the sphincters existing only at the posterior commissure in which the infected anal gland becomes involved following the production of the Pecten band that produces the true posterior commissure fissure.*

The anatomy of the sphincters will be considered first. It is not so simple as is often illustrated. The external sphincter surrounds the internal sphincter both laterally and distally and it is divided into three parts called the subcutaneous, the superficial and the deep. The subcutaneous and deep portions make complete circles. (Fig. 1.) The middle portion, i.e., the superficial, after encircling the anterior and lateral portions of the anus, turns off at a tangent on each side to form a Y, the single leg of which becomes attached to the coccyx.[1,2,3] Between the bilateral legs of the Y, there is a V-shaped space which is called Minor's triangle.[4] (Fig. 1.) It really should be called Minor's triangular or prismatic

space. This space is also called the posterior intermural space. It is bounded on all sides by the sphincter muscles although it can be

was separated from the subcutaneous portion of the external sphincter by an "annular band of fascia" which was
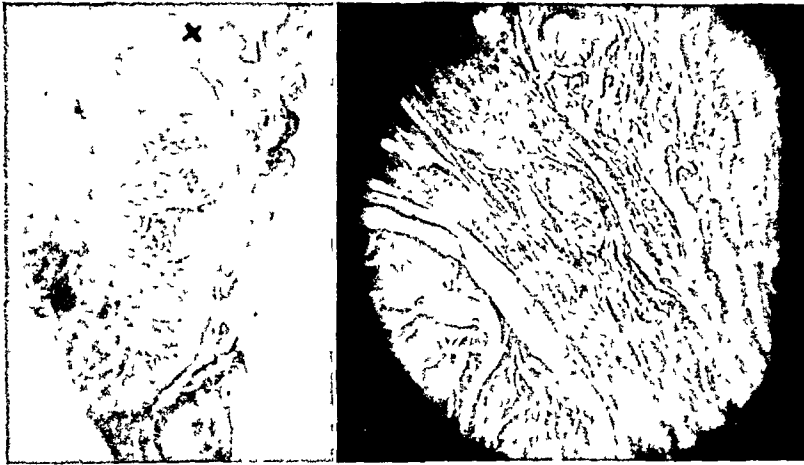
FIG. 2. The left photograph (× 4) illustrates a sagittal section of the subcutaneous portion of the external sphincter at the posterior commissure with some of the longitudinal muscle coming through the intermuscular septum just below ⋋ to become attached to the intra-anal skin opposite. A crypt of Morgagni with the upper portions of an anal duct can be seen at the upper right. In this section, all of the tissues in the latter location are quite markedly contracted and thus pulled down nearer to the external sphincter than they are in the natural state. The right photograph (× 100) shows more in detail this longitudinal muscle seen just below ⋋ in the left photograph.

entered from the anus through the intermuscular septum which is a slit-like window between the internal sphincter and the subcutaneous portion of the external sphincter. It is normally filled with a few decussating fibers of the external sphincter plus some fibromuscular strands from the longitudinal muscular coat of the rectum and connective and areolar tissues. This Y-shaped separation of the superficial external sphincter muscle at the posterior commissure can be definitely palpated, in the normal person, as a depression between the internal sphincter and the subcutaneous portion of the external sphincter.

The Pecten band is a pathological collar of inelastic fibromuscular tissue in the submucosa immediately overlying the intermuscular septum. It was first mentioned by Miles, in 1918, but was more adequately described by Abel[5] in 1932. In 1934, Milligan and Morgan[3] in their admirable description of the anatomy of the anal canal, stated that the internal sphincter

attached to the intra-anal skin at the sulcus between the internal and external hemorrhoidal areas. They called this "band of fascia" the intermuscular fascia. Daniels,[6] in 1936, concluded that Abel's Pecten band and an hypertrophied or fibrosed intermuscular septum were the same thing.

We wish to amplify and correlate all of the foregoing conclusions and observations. Levy[7] as well as Milligan and Morgan[3] have shown how the longitudinal muscle comes down the outside wall of the rectum, ensheaths the various portions of the external sphincter and then becomes attached to the skin at the anal verge and in the intra-anal area at the intermuscular septum. (Figs. 1 and 2.) The fibers of this muscle, in the latter location, take on the characteristics of both smooth muscle and connective tissue, i.e., it is fibromuscular.[3] In the presence of inflammation the connective tissue elements become preponderant to the exclusion of muscular elements especially in the region of the intermuscular septum.

In this way a definitely palpable band of fibrous tissue is produced which is called the Pecten band. (Fig. 3.)

beneath the skin of the anus and emptied by ducts which travel upward and open into these crypts of Morgagni. (Fig. 4.)
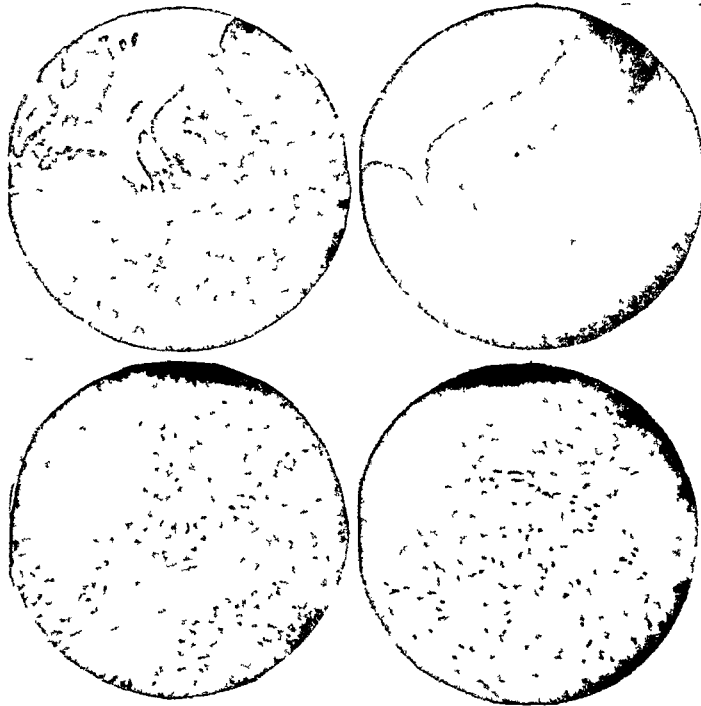
FIG 3 Illustrating the comparison in the character of normal longitudinal muscle at its insertion into the anal skin with what it is when it has been changed into a Pecten band through fibrosis and hypertrophy The upper left photograph (× 100) shows a strand of longitudinal muscle excised during a hemorrhoidectomy The lower left (× 300) depicts more in detail the fibromuscular character of this longitudinal muscle In this section it is quite obviously muscle but sometimes it is hard to say whether it is connective tissue or muscle In contrast, the upper right photograph (× 100) was taken from a Pecten band and the nature of the dense fibrosis and hypertrophy is seen both in this view and in the high power one below it (× 300).

The third provocative or explosive factor in the causation of a posterior fissure-in-ano, is an infected anal gland or "crypt." The closure of the anus is accomplished by the tonic contraction of the two sphincters. This constriction throws the anal skin and mucous membrane into a number of folds between which there are an equal number of depressions, the lower ends of which terminate in cup-shaped pockets called the crypts of Morgagni. These crypts of Morgagni occur at the pectinate or mucocutaneous line which is at the level of the internal sphincter. In many human beings are to be found some little glands lying

These glands have been admirably described and illustrated by Tucker and Hellwig[8] and more recently by M. R. Hill, E. H. Shyrock and F. G. ReBell. They are lined with epithelium and are similar to the para-urethral glands and the glands within the tonsil. As a rule, with some exceptions, we have found them to occur in the adult at twelve, four and eight o'clock, the posterior commissure being twelve o'clock (Blaisdell[1] says five, seven and eleven). It is not uncommon for them to become infected and when this occurs, portions of the epithelium are destroyed, allowing extension of the infection into the surrounding tissues. This

extension is combatted by a walling-off with leucocytes and round cells, the latter being replaced by connective tissue. Subse-

anal glands. Contraction of this band about the duct or neck of such a gland obliterates the latter so that further extension of the
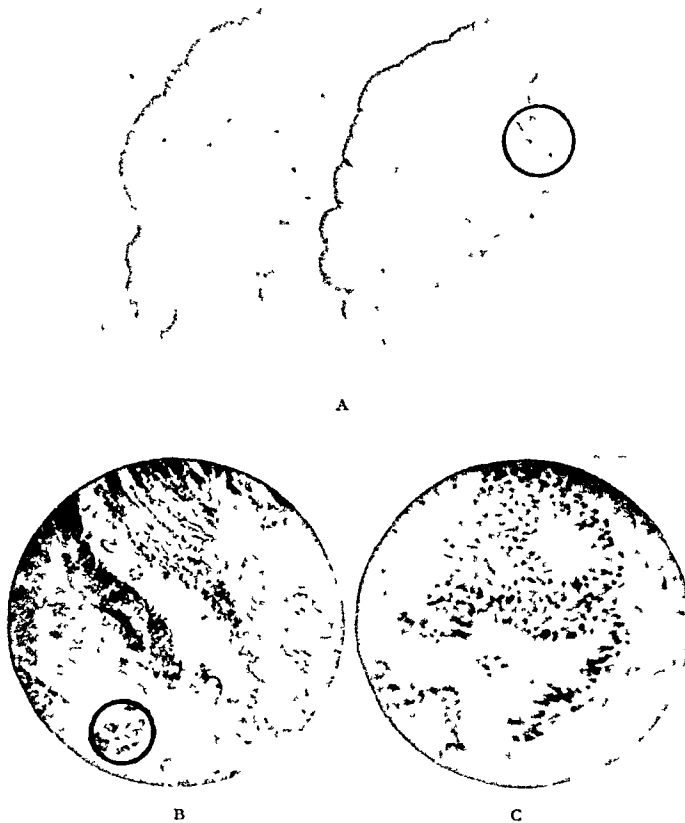
FIG. 4. Illustrating an infected anal duct behind a Pecten band. A, ($\times$ 4) shows two sections of a Pecten band the right one containing (within the circle) part of an anal duct just as it empties into a crypt of Morgagni. The width and density of the fibrosis in this specimen is marked but not extraordinary. The anal duct goes off toward the intermuscular septum and Minor's triangular space at a 45° angle to the anal wall. B, ($\times$ 100) shows a low power view of this same anal duct. Note how racemose it is, also how thin the epithelium is in places (one cell in thickness directly to the right of the circle). C, ($\times$ 300) is a high powered view of that much of which is encircled. In it can be studied in detail the transitional epithelium lining the wall of the duct and the fibrosis surrounding it.

quently, the epithelium regenerates but the reconstructed duct is larger, more racemose and less glandular. Furthermore, it is surrounded by fibrous tissue. This fibrosis involves not only the immediate region of the duct but also stimulates the production of the Pecten band as described above. In fact, it is our belief that a Pecten band only occurs secondarily to infection of these

burrowing is fostered. This burrowing follows the line of least resistance which, except at the posterior commissure, is usually downward between the intra-anal skin and the external sphincter and often the pressure within the duct is great enough to push out the very elastic skin at the anal verge[9,10] so that an anal protrusion is produced which we have called a cryptic

tab.[*,9] However, at the posterior commissure these anal glands either lie with their tips in the intermuscular septum or in at the posterior commissure lies within the fibers of the longitudinal muscle in the intermuscular septum between the two legs
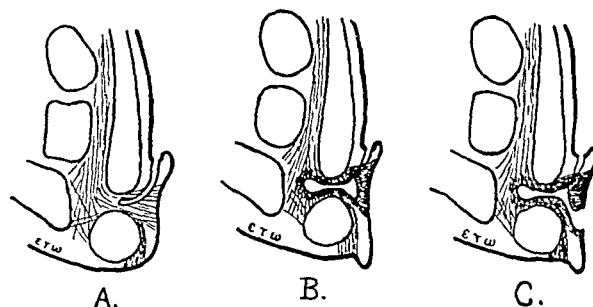
FIG. 5. Diagrams illustrating our conception of the three steps in the pathogenesis of a fissure-in-ano. In (A) an anal duct is shown lying in the intermuscular septum at the posterior commissure. In (B) this anal duct has become infected and has become walled off by fibrous tissue which in turn has strangulated and obliterated the duct in the region of the Pecten band. An abscess burrowing back into Minor's triangular space and down the anal wall is the result. In (c) this abscess has ruptured through a buttonhole opening in the Pecten band and through the anal skin to form the fissure-in-ano (so-called). This diagram is somewhat exaggerated as the abscess does not always extend as far back into Minor's triangular space as is here illustrated. (See Fig. 6.)

Minor's triangular space, or, as they become infected, the line of least resistance is backward toward the latter so that, in either case, infection travels back through the septum toward or into Minor's triangular space (Fig. 4) where it is surrounded on all sides by sphincter muscles except that side filled up by the Pecten band. (Fig. 5.)

It will, therefore, be seen that an infected anal gland at the posterior commissure not only becomes ensnared in the fibrous complications of its own making but also becomes entangled in the peculiar anatomical surroundings existing only in that location. To recapitulate, originally an anal gland

* By common usage these anal glands have somewhat erroneously come to be called "crypts" and when infected the condition has been called "cryptitis." However, even though this nomenclature is somewhat erroneous, as "crypt" and "cryptitis" are shorter and more euphoneous terms than anal gland and anal glanditis, the former will probably continue to be used in preference to the latter. It is not until an anal gland has become enlarged by infection that it can be discovered clinically by entering it with a crypt hook so that when the term "crypt" is used an *infected* anal gland is the entity usually referred to. In contradistinction the crypts of Morgagni are so designated.

of the superficial external sphincter as they go off at a tangent to become attached to the coccyx. Upon becoming infected, it stimulates the fibrosis and hypertrophy of the longitudinal muscle, thereby producing the Pecten band which in turn strangulates its neck. Burrowing takes place but unless escape through Minor's space in the form of a fistula is had, a little abscess is formed surrounded on all sides by sphincter muscles and the Pecten band. (Figs. 5 and 6.) Anything which increases the pressure on this little confined abscess will cause pain and this pressure is decidedly increased by defecation. In fact, during this act, some of the infected material is most certainly squeezed into the sphincter muscles, thereby producing not only the characteristic pain but the spasm as well. The rest of this infected material is suddenly propelled through some small buttonhole rupture in the Pecten band after which it bursts through the very thin intra-anal skin to form the fissure-in-ano. (Figs. 5 and 6.) In several cases we have been able to identify, with a right angled

hook, the little buttonhole opening through the Pecten band in the base of the ulcer from which pus could be expressed. How-

become inelastic and pulled back onto the coccyx. Even if the original abscess and its fissurous opening become completely ob-
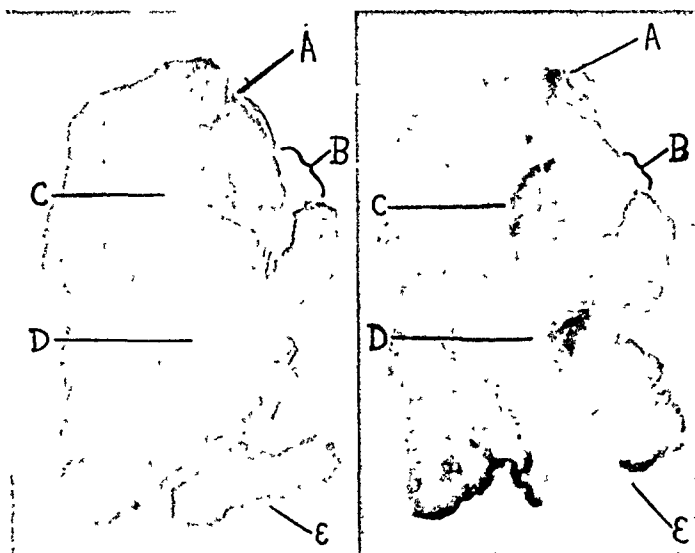
FIG. 6. Photographs of two serial slides selected from a number made from a sagittal section of an old, recurrent fissure-in-ano and its base at the posterior commissure ($\times$ 4). (A) indicates the erstwhile opening of the anal duct. (B) is the "fissure" behind which is a buttonhole opening into the abscess (C). Some extension of the infection down the anal wall is seen at (D) while (E) is the "sentinel pile."

ever, as this opening is very small and as it promptly closes up again, pressure builds up anew in the little abscess only to reproduce the symptoms all over again whenever defecation is once more attempted. Just how far back this little abscess burrows, is hard to say. It probably varies with the individual. In some subacute fissures of weeks' duration, we have found dense, white, indurative scar tissue for half an inch or more back into Minor's triangular space. Occasionally, the typical pain and spasm of a fissure exists but no fissure can be found, in which case the contents of the gland are apparently unable to find any outlet at all. Marked tenderness over Minor's triangular space, however, is present and adequate treatment as for a fissure-in-ano will produce a cure.

If this condition is allowed to become chronic or even to heal spontaneously, more fibrosis occurs, the Pecten band grows thicker and stronger and the sphincters

literated by scar tissue, the seeds for future trouble have been planted, as the intra-anal skin over this fixed fibrosed portion of the anal wall becomes a prey to splitting and abrasion. Its blood supply becomes inadequate and atrophy occurs. Contraction of a Pecten band over the course of many years produces a "contracted anus" not too rarely seen in elderly people.

Several therapeutic procedures designed for alleviation of this type of fissure-in-ano have been evolved but recurrence follows many of them for the reason that the underlying pathological condition has not been removed. The manner whereby each one of these procedures accomplishes its purpose and the reason why recurrence is a frequent sequel may be seen if each procedure is examined in the light of its effect upon the underlying condition: (1) Manual dilatation, under a general anesthetic, increases the drainage from the abscessed gland by temporarily enlarging the button-

hole opening, eliminating the spasm of the sphincters and temporarily loosening or breaking down some of the fibrosis; (2)
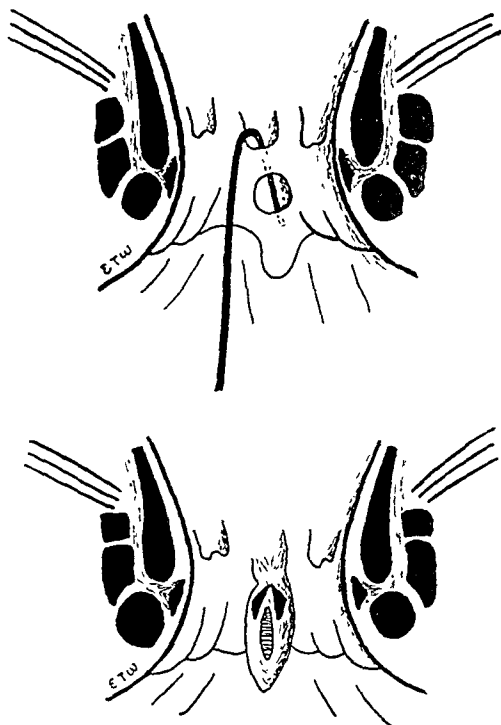
FIG. 7. Diagrams illustrating the two steps in the operation for fissure-in-ano. The upper diagram shows the crypt hook lying in the anal duct behind the Pecten band, behind the "fissure" and in the "sentinel pile." After the hook has been pulled forward, all of the structures on it are excised with the scissors with the result as shown in the lower diagram. Then a posterior sphincterotomy is done whereby the scalpel incises the subcutaneous portion of the external sphincter, the fibers of which can be seen in the base of the excised wound in the lower diagram. The first step has severed most of the Pecten band leaving a few hard fibers at its upper edge. These must be incised when the posterior sphincterotomy is done so that the base of the wound is all soft, muscle tissue. Finally, the skin edges on the buttock are trimmed away. No sutures are used except for ties as drainage and granulation from the bottom is desired.

cauterization unless deep, does no more; (3) an injection of an anesthetic in oil beneath the fissure simply prevents pain and eliminates spasm allowing temporary healing in some cases. However, following none of these methods has obliteration of the gland been accomplished while the fibrosis and the Pecten band still exist or are soon reformed. (4) Excision of the fissure will be successful only if this excision happens to include some or all of the gland or if some or all of the Pecten band is accidently incised. (5) Incision of the Pecten band, either through the base of the fissure or elsewhere, is theoretically promising but we personally have had recurrences even following this operation. Apparently both the gland and the Pecten band regenerated.

It will, therefore, be seen that in order to secure a radical and permanent cure, three ends must be achieved, namely, as much of the anal gland must be excised as is possible; the Pecten band must be completely incised and its regeneration prevented by separation of the severed parts long enough for resolution to begin and adequate drainage of the intermuscular septum at the posterior commissure and of Minor's triangular space must be obtained and maintained until granulations eradicate any vestiges of crypt remaining in either of those regions.

Two fairly simple operative procedures done under novocaine and oil anesthesia will accomplish the desired results. The first one is a cryptectomy whereby the anal duct together with the papillas, fissure and the "sentinel pile" are picked up on a crypt hook and excised in one piece. (Fig. 7.) As the duct runs down through or beneath the Pecten band, this procedure will of necessity also partially incise the latter. This eliminates much of the underlying pathological condition except what is back in Minor's triangular space. Then in order to obtain adequate drainage of the latter, a posterior sphincterotomy is performed such as has been advocated by Mulligan and Morgan,[3] Blaisdell,[1] Gorsch[11] and Daniels.[6] In this operation, the whole of the subcutaneous portion of the external sphincter is incised at the posterior commissure together with any remaining fibers of the Pecten band. This posterior sphincterotomy is very important. Not to do it is to court either recurrence of the fissure or production of a blind fistula[1] going back at

a right angle through the intermuscular septum into Minor's triangular space. Finally, all overlapping edges of skin are cut back, a penrose drain is inserted and a pressure bandage applied. The drain is removed in twenty-four hours. A few days of hospitalization is all that is usually necessary. The sphincter ends become reapposed fairly rapidly and full healing takes place in from two to four weeks depending upon the amount of skin excised. In an early acute fissure with little fibrosis only a small amount of skin need be removed, but in an old or recurrent case with a thick Pecten band, wide excision of skin will prevent healing long enough for resolution of the fibrosis to take place and a slightly funnel-shaped anus (from without, in) will result. The indirect effects of this operation are often remarkable. The anxious appearance vanishes, color returns to the cheeks, vague reflex aches and pains disappear, cheerfulness and calm succeed fault-finding and irritability and in many cases a long standing constipation is corrected.

SUMMARY

The syndrome of acute symptoms accompanying a true fissure-in-ano at the posterior commissure has long called for a logical and scientific explanation but it was not until the last few years that the presence of infected anal glands and the Pecten band was recognized and the intricate anatomy of the anal sphincters is still a subject for study.

It is shown how all three of these factors are involved in the genesis of a fissure-in-ano. An anal gland becomes a prey to infection which ultimately is combatted by the production of fibrous tissue especially in the neighborhood of the fibro-muscular longitudinal muscle. Fibrosis of the latter results in a Pecten band which strangulates the duct or neck of the gland thereby producing an intersphincteric abscess. Rupture of the latter through the anal wall creates a round or oval ulcer which when the anus is not dilated, looks like a fissure or crack.

Logical treatment requires elimination of the underlying causative condition. This is accomplished by excision of the infected anal gland, incision of the Pecten band and adequate drainage of the abscess by means of a posterior sphincterotomy.

REFERENCES

1. Blaisell, P. C. Pathogenesis of anal fissure and implications as to treatment. *Surg., Gynec. & Obst.*, 65: 672–677, 1937.
2. Morgan, J. W. Pectenosis and minor maladies of the anal region. *Surg., Gynec. & Obst.*, 59: 806–809, 1934.
3. Milligan, E. T. C. and Morgan, C. N. Surgical anatomy of the anal canal. *Lancet*, 2: 1150, 1934.
4. Minor, W. C. Proctotomy with implantation for contraction of anal canal. *Med. Herald*, 46: 155–160, 1928.
5. Abel, A. L. The pecten: the pecten band: pectenosis and pectenotomy. *Lancet*, 1: 714–817, 1932.
6. Daniels, E. A. Anal fissure, anal spasm and anal stenosis. *Am. J. Digest. Dis. & Nutrition*, 3: 775–783, 1936.
7. Levy, E. Anorectal musculature. *Am. J. Surg.*, 34: 141–198, 1936.
8. Tucker, C. C. and Hellwig, C. A. Histopathology of the anal crypts. *Tr. Am. Proc. Soc.*, 47–52. 1933.
9. Whitney, E. T. and Keane, J. F. Perianal cryptic tabs. *New England J. Med.*, 221: 303–306, 1939.
10. Drueck, C. Cryptitis. *Illinois Med. J.*, 71: 528–531, 1937.
11. Gorsch, R. V. Fissure-in-ano, its pathogenesis and treatment. *Med. World*, 56: 300–320, 1938.

PILONIDAL CYST*

REPORT OF A NEW PROCEDURE FOR OPERATION AND TREATMENT

FIRST LIEUT. DAVID BREZIN, M.C.

Chief of Septic Surgical Section, Station Hospital

FORT BRAGG, NORTH CAROLINA

WE believe we have produced a procedure for operation and treatment of pilonidal cyst which has materially reduced the healing time. The purpose of this paper is to present experiences gained in the treatment of thirty patients. The study was undertaken because previous methods were not satisfactory for use in the military service.

In the military service the problem of pilonidal cyst is a prominent one. Following operation it is the general policy that the soldier shall not be returned to duty until convalescence is complete. For example, the soldier cannot do duty while he has an open granulating wound and return to the dispensary for periodic dressings. Therefore, the surgeon is confronted with the maintenance of the patient in the hospital until the wound is solidly healed.

The incidence of pilonidal cysts is high in the army. This may be expected because the recruits are in the proper age group and incidence is higher in males. Mild to moderate trauma is inherent in the occupation and exigencies of modern military methods. The type of trauma may range from that gotten from vigorous calisthenics to direct injury incurred from riding over rough terrain in trucks. Trauma as a predecessor of infection draws attention to the presence of a cyst. In this series moderate to severe infection was present in all cysts operated upon. During the First Army Maneuvers in the Carolinas, 33 per cent of the admissions to the Septic Surgical Service at this hospital were actually pilonidal abscesses. Often the cavities had been extended by infection beyond the confines of the cyst wall and over the buttocks. The methods previously used were variations of block excision. They were: (1) block excision packed open to granulate in; (2) block excision with mattress suture closure with sulfanilamide powder in the wound; or (3) closure where various types of relaxing incisions had been made to close the wound without tension. None of these methods was entirely satisfactory primarily because of the prolonged healing time and also because of complications which were inherent in healing of a scar in the midline crease.

Consequently, in order to establish a procedure which would be more ideal we set down the following aims: First, we attempted to devise a method by which we could be more certain of excising all the abnormal and infected tissue. This meant a procedure by which a more anatomical dissection could be done with better visualization of the sac structure. Second, we sought for a method to facilitate healing and reduce hospitalization to a minimum. In general to accomplish this end meant finding a substance which would not only reduce the possibility of infection but would also stimulate healing. Third, we endeavored to leave the soldier with a solidly healed scar which would be least likely to break down. We believed that this latter aim could best be accomplished by reducing the need for new epithelium by retaining the original thick skin.

EXCISION

Block excision by sharp dissection or with cautery, with or without closure, has been the most popular procedure. This type of excision is a blind method which

sacrifices a great deal of surrounding normal tissue. Likewise because of the rigorously predetermined elliptical type of

shall cross the midline crease. For this reason there can be no standard incision. Often because of the number and the
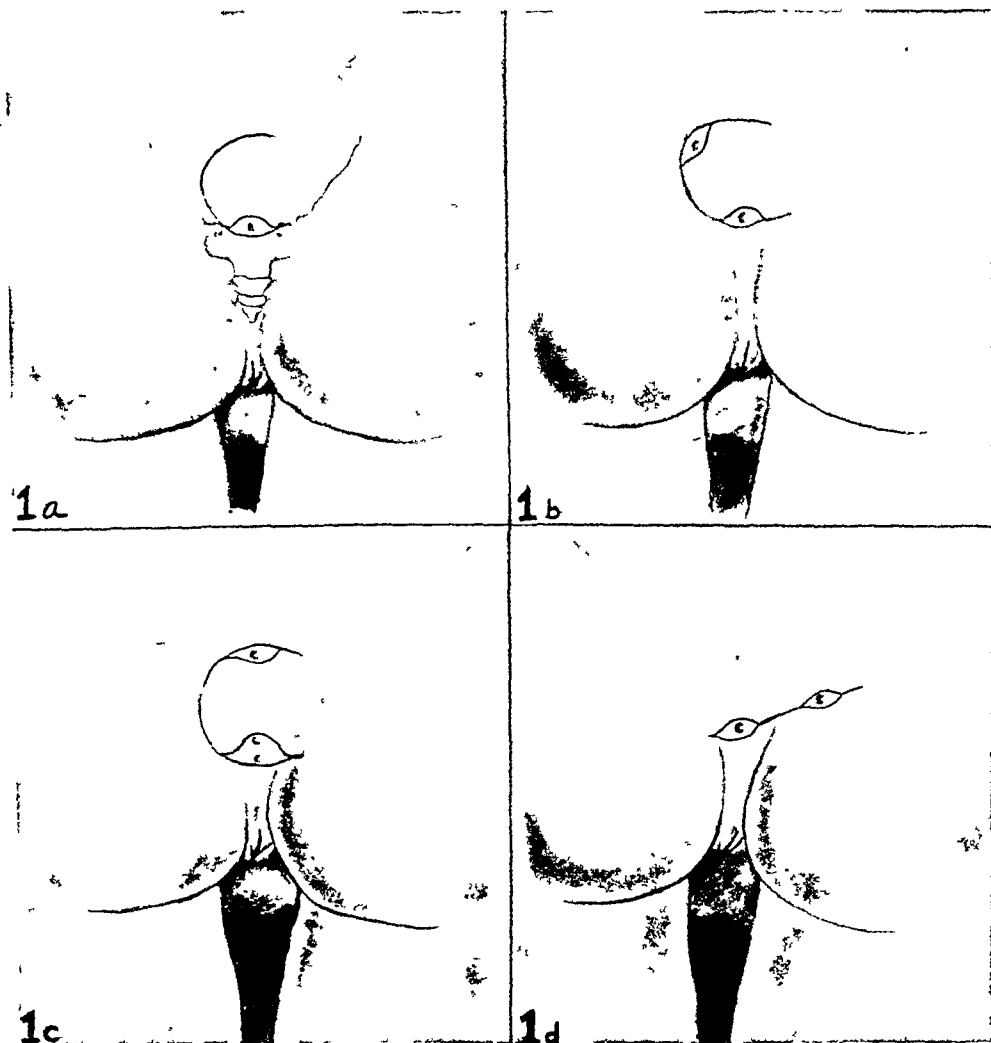
FIG. 1. a, b, c, d, the relative positions and arrangement of the sinus openings are portrayed. The heavy black line indicates the line of incision and outlines the skin islands left surrounding the sinus openings.

incision, cyst wall and infected scar which may extend laterally or down to the anal wall, may be transected and left in the depths of the wound. Not only may there be a large percentage of recurrences but also absence of healing.

The Incision. Because healing and epithelization, owing to tension and maceration, are so often prolonged in the midline, the primary precept for the incision is that just as little of it as possible shall be in, or

arrangement of the sinus openings the ingenuity of the surgeon is taxed. One can always depend on a midline sinus opening. However, additional sinus openings which we shall designate henceforth as accessory sinus openings may be present either in the midline or to one side of it. The midline sinuses may be close together, two or three in number, or they may be spread apart. Accessory openings lateral to the midline are usually found in the skin over the left

buttock. These are the result of an extension of the infection when midline openings have become obliterated through one

FIG 2 The pedicled flap is reflected and the cyst is exposed. The skin islands surround the sinus openings and are dissected out attached to the cyst.

means or another. However, the accessory sinus may be present anywhere within a radius up to 1½ inches from the original midline opening. The original midline opening is usually found at the level of the sacrococcygeal joint. (Fig. 1, a, b, c, and d.)

At the operating table all sinus openings are carefully probed and the direction in which the probe passes is noted. This is very important since it alone determines the type and direction of the incision. A mixture of methylene blue and hydrogen peroxide is injected into any of the patent openings in order to stain the tracts for recognition during dissection. In the course of the study it became apparent that one type of incision was more frequently adaptable and efficacious than others. (Fig. 1, a, b and c.) This type begins and ends on the same side of the midline and is U-shaped. It circumscribes small elliptical pieces of skin which are left about the sinus openings. Minor variations are necessary depending upon the circumstances but this type of incision permits the reflection of a

pedicled skin flap. After reflection, the exposure afforded is good not only for tracts running longitudinally in the midline but also for those which have sacculations to one side, or which may extend down and attach to the anal wall. The pedicled flap likewise saves the original thick skin for closure.

A second type of incision is usually transverse or oblique and is adaptable for sinuses or sacs which have accessory sinus openings to either side of the midline more or less at the same level of the original midline sinus opening. Elliptical pieces of skin are likewise left about the sinus openings when this type of incision is utilized. Here, however, instead of a pedicled flap the skin edges are undercut from ¼ to ½ inches on both sides, and turned back. This usually affords sufficient exposure and permits easier dissection. (Fig. 1d.)

The Dissection. When the U-shaped incision is employed the skin edges which form the border of the skin flap are grasped in Allis forceps and tension upward is applied. Dissection to reflect the flap is started at the immediate junction of skin and subjacent subcutaneous tissue. The flap is then carefully undercut. Often the cyst wall is immediately underneath the skin and the surgeon must cut close to or even split skin in order to avoid entering the sac. Methylene blue aids materially in defining the boundaries but even without stains the wall stands out sharply as a smooth opaque shiny structure. If the sac is entered, any cyst wall tissue left on the undersurface of the flap must be excised. After reflecting the flap and undercutting the opposite skin edge for ¼ inch the excision of the cyst is begun. (Fig. 2.) When the transverse or oblique incision is used, the skin edges are undercut for ¼ to ½ inch in the same manner as the flap. In either case a probe is inserted into the sinus opening into which it passes most easily and kept in place until the dissection has begun. The end of the probe denotes the lateral and upper margin of the cyst wall and indicates where separation from

the normal tissue should begin. The most cephalad skin island around the sinus opening is grasped in an Allis forcep, tension upward is applied and the tissue at the lateral margin of the cyst wall is incised and the wall thus partially freed. A plane of cleavage is defined very shortly; steady tension applied to the sac makes the fibrous bands attached to the cyst wall stand out sharply. These bands are transected. The sac may be further separated by inserting a Mayo scissors laterally and spreading the blades. Where the sac wall is particularly thick, blunt separation with gauze over the forefinger very often materially aids in removal by shelling out the sac in the correct plane of cleavage.

Although the sac or sinus usually originates as a midline structure, its eventual outlines have been found to be quite diverse. We have found prolongations into the fatty tissue above the gluteal muscles. Likewise large sac-like prolongations have been found to extend downward onto the anal wall and to be subjacent to the skin lateral to the anal opening. The depth of the sac is also extremely variable. It may be entirely superficial and following its removal only a small midline defect will be left in the subcutaneous fat. In other instances it may run downward and part of the cyst wall will be adjacent to the sacrococcygeal ligament.

It would appear entirely logical then that block excision using a set elliptical incision is the type of operation in which abnormal tissue more likely would be transected and left in the wound. On the other hand, by tracing the sac boundaries, by what we term anatomical dissection, the chances of removal of the entire sac and infected scar tissue would appear to be increased infinitely. As an instance in which this is graphically portrayed, we overlooked a small midline sinus opening hardly larger than a normal skin pore. The U-shaped incision was used in this case and while the skin flap was being reflected a small gelatinous sinus tract about 2 mm. in diameter was found leading

into the above mentioned sinus opening. Recognition meant effective excision. It goes without saying that in order to accomplish a well planned anatomical dissection extreme care must be paid to hemostasis. Visualization of the field is imperative at all times.

HEALING

Closure. Our cases fall into two classes: those which could be closed entirely and those in which only partial closure could be accomplished. There has been a diversity of opinion concerning any closure. The chief objection has been that the area is always potentially infected, and as such one invites actual postoperative infection by closure. Complete closure with primary healing is the most ideal procedure following removal. We have combatted effectively the inclusion of potential infection by the use of a sulfathiazole-cod-liver-oil ointment. We have used this ointment in the wounds of severe hand-space infections, in the musculature and skin following thoracotomy for empyema and in the muscular layers following drainage of a perinephric abscess in which the layers were sutured. Infection cleared and healing occurred in a most dramatic manner.

The ointment is composed of sulfathiazole powder 10 per cent, cod liver oil 10 per cent in a lanolin base.

When this study was planned originally the procedure outlined was to close the skin flap partially and permit the wound to heal as the perineal wound following abdominal perineal resection of the rectum. Packing permeated with the ointment was placed underneath the flap. Postoperative dressings revealed the wound to be extremely clean, without odor and a minimum of drainage. Aerobic and anaerobic cultures from the depths of the wound were taken and bacterial contamination was found to be either absent or the colonies at a minimum. In the case in which bacteria were present the morphology was so altered that the bacteria could not be identified and it was evident that severe

attenuation had occurred. Further bacteriologic study indicated that the ointment had bacteriostatic properties only
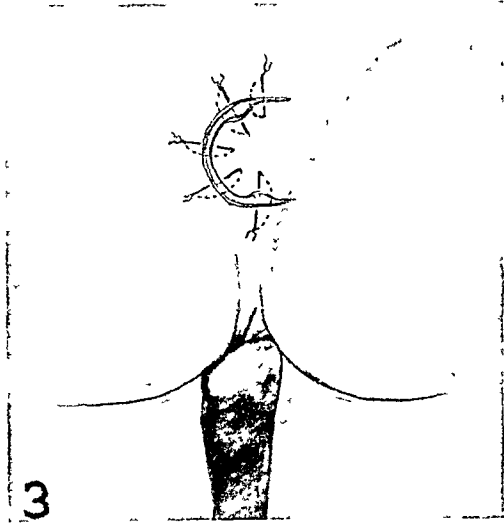
FIG. 3. The flap is replaced and silkworm Stewart sutures are placed ready for tying.

on interaction with the body tissues. Control studies with the ointment used in culture medium did not reveal any bacteriostatic properties at all. With the use of the ointment the logic for primary closure, complete or incomplete, became apparent.

Primary Closure. Most of the incisions may be closed completely. The type of excision does not sacrifice normal tissue and the subcutaneous structures usually may be brought together without tension. Plain No. o catgut sutures are used to close the defect. Suture bites about ½ inch in length are taken on either side and if the sacrococcygeal ligament has been exposed by dissection, the suture is drawn through this structure as a mattress stitch, affording additional anchorage. Experience has shown that all sutures should be placed before the ointment is instilled in the wound. A little careful planning will usually permit the tissues to be drawn together so that the opposing sides fit without tension. The ointment is then placed in the cavity and the sutures are tied. Closure squeezes out the excess ointment and during healing the remainder of the

oily substance slowly leaks out after it has reached body temperature. The skin flap or skin edges as the case may be, is replaced and approximated with silkworm Stewart sutures. (Fig. 3.) Sometimes the flap must be revised in order to fit the opposite side. This may involve trimming away redundant skin or stretching skin edges slightly to fit in the upper and lower angles. Usually a slight midline depression into which the skin flap falls, takes up any redundancy bringing the edges into perfect approximation. Extreme care must be used in approximating the skin edges because postoperative motion may pull the flap so that one skin edge may override the other.

Partial Primary Closure. In cases in which the defect cannot be drawn together at all points without tension, part of the defect is left unsutured. We have had no case in which the angles of the defect cannot be approximated, so the eventual dead space is left in the center and is considerably smaller than the open granulating wound following block excision. The residual space is filled with the ointment and the skin flap reapplied as in the primary closure except for the suturing. Part of the skin is left open at one or the other side of the midline so that the ointment may escape as granulation occurs from the bottom and so that ointment may be inserted daily. The healing time has been decreased in these cases not only because of the decrease in the size of the defect, but also because the ointment has shown itself to be extremely stimulating to the growth of granulations and epithelium.

In two cases in the series the cyst wall extended down to the subcutaneous tissues to the right of the anus. These cases were primarily closed except for a ½ inch stab wound in the skin to the right of the anus into which a small rubber drain was placed. The excess ointment found egress from the depths of the wound through the stab opening and healing was by primary intention.

Pre- and Postoperative Care. These cases were all infected, some mildly some se-

verely. Preoperative drainage was encouraged if possible through the original sinus openings which were probed open. The patient received hot sitz baths several times daily until inflammation cleared; no specific time interval was allowed preoperatively. In most instances the sitz bath régime lasted only a few days.

An enema was given the night before operation. Preoperative medication was a grain and a half of seconal one hour before operation, morphine sulfate gr. $\frac{1}{4}$ and atropine gr. $\frac{1}{150}$ one-half hour before.

At the operating table spinal novocain 100 mg. was used for anesthesia. The patient was placed on his stomach, the buttocks held apart by adhesive straps, stuck to the side of the operating table. The field was prepared by cleaning with ether and painting with tincture of merthiolate.

Following operation one ounce of mineral oil was given daily for four days. Bathroom privileges were permitted but the patient was otherwise restricted to bed for four to five days. Postoperative medication for pain was almost entirely unnecessary because of the soothing properties of the ointment. In the cases in which the incision had been completely closed, unless there was a temperature elevation, the first dressing was done on the seventh postoperative day at which time the sutures were removed and a sulfathiazole-cod-liver-oil ointment dressing reapplied. In the cases in which the incision had been left partially open the first dressing was done on the third postoperative day when more ointment was instilled in the dead space through the slitlike opening in the skin. We found the subsequent daily instillation of the ointment could be accomplished more effectively by injecting it through a metal ear syringe. Sutures were removed on the seventh postoperative day. In these latter cases it was found that granulation often heaped through the skin defect or that the skin edges turned down. Intervention was found unnecessary since nature revised the wounds in all cases. Granulation shrunk back into the wound and the skin edges were bridged over by smooth epithelial tissue.

RESULTS

In comparison with other methods the healing time following excision of pilonidal cysts by this method has been markedly reduced. The average healing time in thirty cases, including those of primary closure and of partial primary closure was twenty-six days. The range in healing time is from ten to forty-three days. Forty per cent of the series were healed and discharged from the hospital in from ten to twenty days; 30 per cent in from twenty to thirty days, and 30 per cent in from thirty to forty-three days.

In 50 per cent of the cases in which complete primary closure was effected, following removal of the sutures on the seventh postoperative day, the skin edges at the lower angle of the incision separated for $\frac{1}{4}$ to $\frac{1}{2}$ inch. This always occurred where the incision crossed the midline which further substantiates the rationale for using an incision which does not necessitate as much midline healing as is necessary in the block excision. The particular stress exerted on the skin edges at this point is the same as that exerted on the granulation and epithelium throughout the entire course of the midline following the old types of excision. Retention of sutures for a longer period of time, or adhesive strapping which kept the buttocks from spreading, tended to avoid this separation. However, when sutures are retained for more than seven days they show a marked tendency to cut through the skin flap. When separation does occur, the small size of the midline aperture, its transverse position, and the fact that the ointment not only stimulates healing but also keeps the wound free of infection, make subsequent healing a matter of a short additional time. There was one case of gross wound infection in a patient who developed generalized furunculosis two days after operation. The wound in this case was opened widely and packed with the oint-

ment daily. Healing occurred in thirty-six days.

The healed wounds have remained solidly healed following discharge from the hospital. There has been no instance in which splitting of the incision has occurred; there has been no superficial maceration, no abscess formation and no tenderness or pain. Sufficient time has not elapsed so that an evaluation of end results can be made, although immediate results have proved to be very good. In a second series of cases which we have started the results appear even more promising.

SUMMARY

1. A new type of operation and treatment for excision of pilonidal cyst is presented. We argue that the chances for complete removal of cyst and infected tissue are increased by an "anatomical dissection."

2. A new type of incision avoids the midline except where it crosses it in the transverse direction.

3. The method used retains original skin, sacrifices little normal tissue and the defect is closed partially or completely.

4. Healing is stimulated and the possibility for infection is reduced by instilling a sulfathiazole-cod-liver-oil-lanolin ointment in the wound previous to closure. This substance is also used for subsequent dressings.

5. Healing time is markedly reduced by decreasing the necessity for granulation and by retention of original skin.

6. Immediate results indicate a solidly healed wound and absence of complications attendant upon midline healing.

REFERENCES

1. KOOISTRA, H. P. Pilonidal sinuses. Am. J. Surg., 55: 3–17, 1942.
2. ROGERS, H. and DWIGHT, R. W. Pilonidal sinus. Ann. Surg., 107: 400–418, 1938.

SKELETAL FIXATION OF FRACTURES

H. H. HAYNES, M.D.

Chief of Staff, St. Mary's Hospital

CLARKSBURG, WEST VIRGINIA

SINCE the origin of man, broken bones have been one of his major problems.

Recent facts brought to light show that the early Egyptians practised the treatment of fractures by internal and external fixation. More or less crude applications of these principles were used until the seventeenth and eighteenth centuries. It is from this period that the evolution of our modern concepts of rational treatment began. The early bone setters reached their zenith with Hugh Owen Thomas in the latter part of the nineteenth century.

At this time treatment of fractures by fixation and rest was generally accepted, and there were in vogue several procedures to achieve this end. The French school made use of the plaster of Paris cast and splint, while another large group of contemporaries subscribed to the principle of fixation by extension as advocated by Davis, Sayre and Taylor of New York. Still a third large group, the disciples of Hugh Owen Thomas, invariably practised the doctrine of "enforced, uninterrupted and prolonged rest." Thomas early in his career recognized the disadvantages of plaster casts. He devised an instrument to produce fixation and rest without constriction. This instrument known as the Thomas splint is widely used today. The fact that limited and controlled motion at the site of fracture produced excess callous formation was recognized early by Thomas and led him to the practice of percussion, at frequent intervals, over the site of fracture.

At the time of Lucas-Championniere, treatment by immobilization was considered of prime importance. Lucas-Championniere swung the pendulum in the opposite direction and convinced many that motion was of prime importance in the treatment of fractures. Thirty years later the medical profession came to the conclusion that had been arrived at over one hundred years before by Jean Pierre David who said that "rest and motion had respective and equally important places in the rational treatment of fractures."

Kellogg Speed[1] in his textbook says the following:

"The development of the distraction of nail extension is interesting. In 1903 Codivilla first applied a cast to a fractured leg from the toe to the pelvis. The next day he cut the cast circularly, dividing it about the middle of the thigh. By means of strong traction the severed surfaces of the cast were separated, and the space created was filled in with fresh plaster of paris, traction being maintained until setting. This procedure could be repeated. The method was abandoned because decubitus sores developed about the tuber ischii and dorsum of foot.

"To avoid these pressure ulcers Codivilla left the foot and ankle free and put two lateral irons into the lower end of the cast. The free ends of the iron ran down beside the ankle and were fitted by means of holes over a nail driven through the os calcis. Pads were applied over the ischium and decubitus was avoided.

"Steinman, in 1907, discarded the cast altogether and applied traction directly to the nail ends passing through the os calcis.

"In Von Eiselberg's Clinic in 1901 Kafer, in putting a cast on a leg, incorporated a turnbuckle on either side. The cast was cut and the halves distracted by turning up the turnbuckles.

"Hackenbruch[2] improved this method by putting a ball and socket joint at either end of the turnbuckle where it joined onto the

embedded plate. By distraction, shortening in the leg was overcome inside of forty-eight hours; then the four ball-and-socket
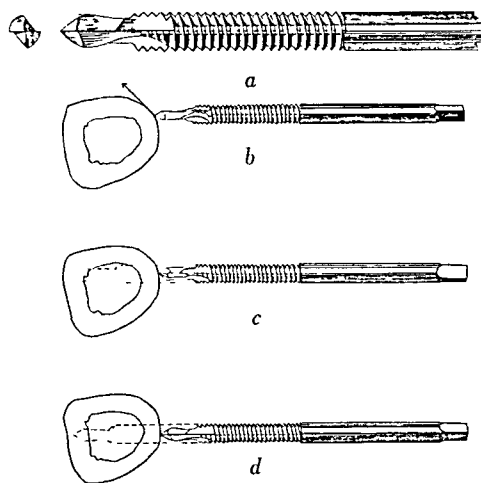
FIG. 1. a, combination drilling and self tapping half pin. b, poor site of insertion, outer one third of diameter of bone; c, fair site of insertion; d, preferable site of insertion.
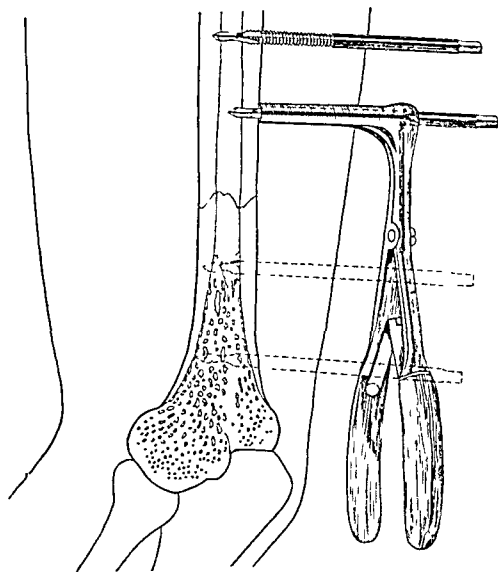
FIG. 2. To prevent soft tissue trauma, the bivalved sleeve is placed around the pin, worked down to the bone surface and held there while the pin is inserted into the cortex.

joints were cautiously loosened, and any lateral displacement was corrected.

"The reduction was checked by roentgenograms, and when a satisfactory adjustment was reached all points were firmly

fastened and the patient was allowed to walk on the leg, thus held in perfect position. Patterson's method[3] is the same. Gerster[4] has adopted Steinman's nail extension as a part of a splint for maintaining extension during transportation. To avoid the pressure on the ischium he used an upper padded ring made in two halves hinged behind and locking in front.

"The splint was composed of long bars of hard wood, which do not interfere with roentgenograms, and the lower end was formed by a stirrup, with slotted bars permitting adjustment by thumb-screws. Tongs were applied to the nail, and a cross bar held them up, while a rope attached them to the distal end of the splint."

Arbuthnot Lane, in 1894, developed internal fixation of fractures by the use of metallic bone plates. F. H. Albee, in 1911, first introduced autogenous bone grafts for internal fixation.

The basis of the modern rational approach to the treatment of fractures is that of anatomical reduction. Anatomical reduction means the restoration of the normal axis of the extremity by proper alignment, with consequent protection of joint movement from abnormal strain and the preservation of unaltered lines of contraction for the adjacent muscles.

The idea of skeletal fixation was conceived by the author while working with the late Colonel Joseph Blake at Red Cross Hospital No. 2, at Paris, in 1918. Not being able to work out a satisfactory connection with sufficient flexibility and strength, nothing was done until 1932, when a practical bar was made up.

From this date it took five years to interest any surgical instrument manufacturer sufficiently to produce the splint. Early in 1938, the first splints were made, none of which were applied until June, 1938.*

Doctor Harold R. Conn,[5] of Akron, Ohio, produced a splint which he reported. This has two pins in each fragment which

* Manufactured originally by the Zimmer Mfg. Co., Warsaw, Indiana.

come through the skin and has an adjustable metal support. This is much lighter and much less flexible than the splint we are describing.

of Philadelphia, developed a splint very similar in principle which he uses in the veterinary field.

The method of treatment of fractures
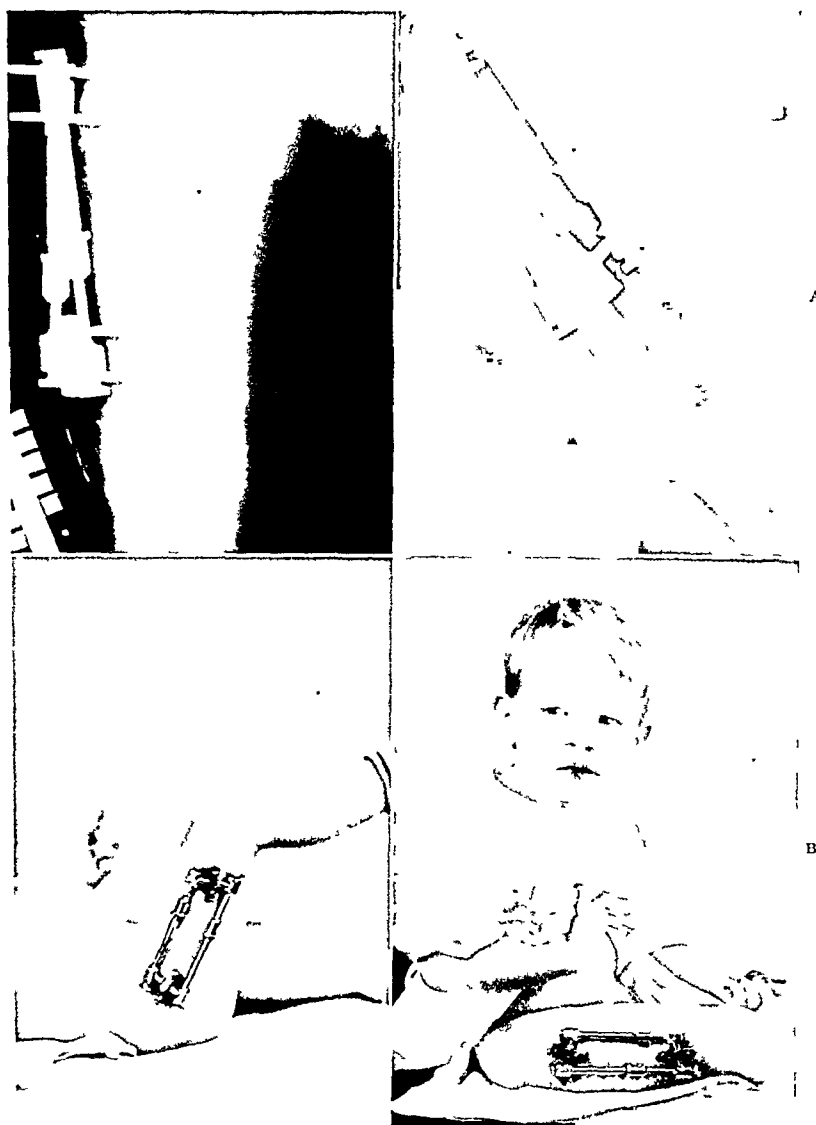
FIG. 3. A, anteroposterior and lateral x-ray plates (splint applied). B, showing radius splint applied to the baby's thigh and the amount of freedom of mobility of joints.

Doctor Rogers Anderson[6] uses skeletal fixation with his reduction apparatus, supported by plaster cast which he also reported in 1938. Doctor Anderson has since designed and is now using a skeletal fixation apparatus with a metal support.

Doctor Otto Stader, veterinary surgeon,

described in this paper achieves anatomical reduction by the use of an instrument combining the principles of internal and external fixation without the necessity of open operation. Fixation is limited to the individual segment except in those cases in which the line of fracture is closely adjacent

to or invades the joint proper. With this instrument fixation of the highest degree is obtained for the individual segment and

rate picture of the process either with words or illustrations. The size and shape of the splints vary markedly according to
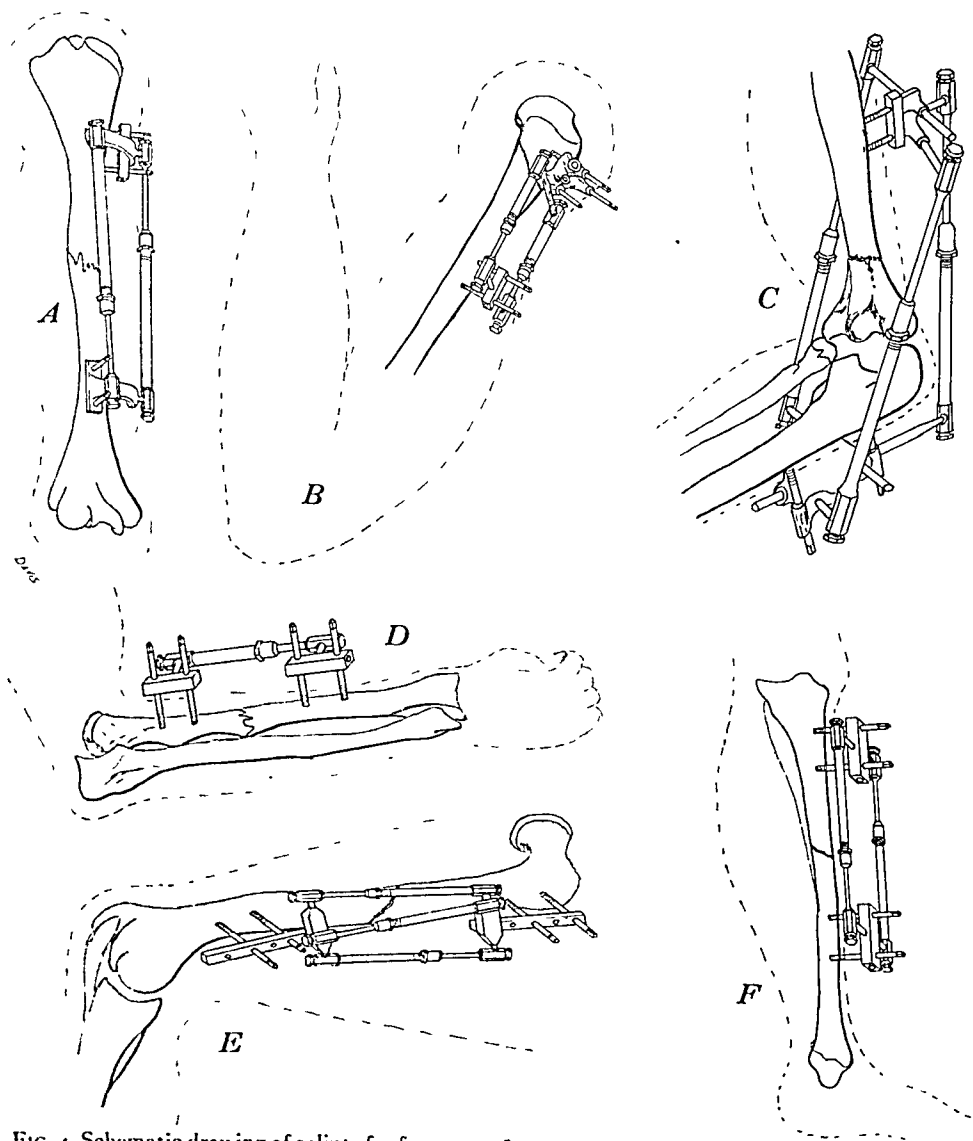
FIG. 4. Schematic drawing of splints for fractures of surgical neck, shaft and lower end of humerus, radius, tibia and fibula.

the important motion of the entire member is preserved.

This method of treating fractures is in the early developmental stage and is necessarily crude. Time and experience are certain to bring great improvement.

In describing a new method of treating fractures, in which so many principles are involved, it is difficult to convey an accu-

size, shape and function of the bone or bones involved. However, the same principle of skeletal fixation of the individual bone holds good throughout. The principle underlying this method of treatment is to anchor a strong pier to each distal fragment and bridge the fracture or fractures with a fabricated steel network. In this manner the distal ends of the bone are maintained

in definite relation to each other, which enables them to function as they did before the fracture. This principle is carried out bearing connecting unit, which together compose the anchoring pier and the fabricated bridging device. (Fig. 4.)
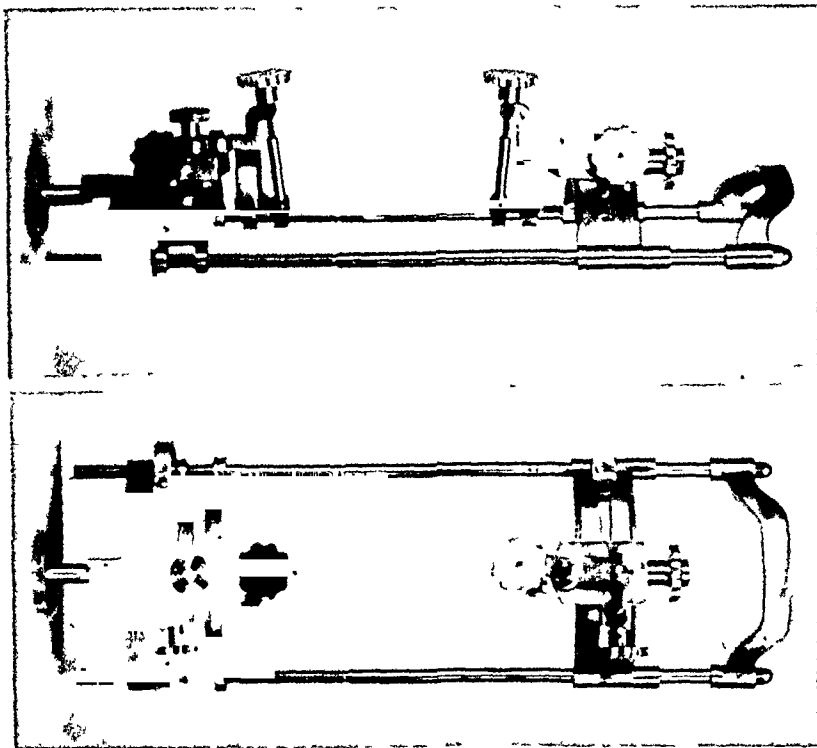
FIG. 5. Top and side views of reduction apparatus. Extension apparatus has triple-compounded leverage for the extension and motion in three planes, all controlled by gears in order to prevent slipping, jerking or losing apposition. After satisfactory reduction is made, the bars are applied and the reduction apparatus is removed.

in fractures of all the long bones except in those instances in which, either on account of lack of distance or comminution, adequate application of the anchoring pier cannot be made.

Skeletal fixation of joints is made use of in the following conditions: intra-articular fractures in which motion of the joint is undesirable, and fractures in which the distance is inadequate or the comminution too great for proper application of the anchoring pier. Under these conditions, a modified appliance is used to bridge and fix the joint, but the underlying principle is the same as that used in fixation of the individual bone.

The appliance is composed of three functional units: They are the two, or more, attaching pins and the intermediate, stress-

Solid attachment to the bone is secured by the use of either threaded half pins or through-and-through pins. (Figs. 1 and 2.) The half pins are made of stainless steel, with a cutting point, deep threads and a head for adaptation to a chuck wrench or brace. The half pins vary in length from one inch to four and one-half inches, and in diameter from three to seven thirty-seconds of an inch. The through-and-through pins are the usual type of Steinman pins.

The stainless steel blocks are composed of rectangular bars of varying sizes, containing multiple holes which accurately fit the threaded half pins they are made to accommodate. The pins are secured in the block by set screws. The bridging device is firmly attached to the blocks by means of

the multiple spheres, which are connected to the blocks by slender shanks. The appliance is demonstrated in Figure 4.

bars with a split sleeve adjustment. On each end of the telescoping bar is a friction clamp with a universal joint for

FIG. 6. Pins in place eleven months in comminuted fracture of the tibia and fracture of the lower end of the fibula. "The alignment is very satisfactory. There is evidence of considerable new bone formation at the site of the fracture of the tibia, but none is seen in the region of the fracture of the fibula. The most interesting finding is the lack of bone absorption around the screws in the traction device. Minimal absorption is noted around only one of the three visible screws. No bone absorption is observed around either of the others. This is unusual after an interval of eleven months." (Comment by Whitmer B. Firor, M. D., Baltimore, Md.)

When the through-and-through pins are used, it is necessary to grasp firmly both ends of both pins. This is readily accomplished by a yoke. The attachment of the yoke to the pins is accomplished by collars, built in the yokes. These collars grasp the ends of the pins and are in turn secured by set screws. The collar attachment of one pin of each pair of pins is flexibly adjustable to facilitate application of the yoke. Multiple spheres mounted on slender shanks attached to the yoke provide for connecting the bridging device.

Fixation is accomplished by a fabricated bridging device composed of telescoping

grasping the spheres mounted on the block or yoke.

In the experience of the author this treatment has been quite satisfactory in the following cases: simple or compound fractures of the long bones and mandible; as a substitute for bone plating; as a support in the after-treatment of bone grafting and correction of angulation and malposition. Judging from my military and subsequent experience in traumatic surgery, and the report of Dr. Phillip D. Wilson and W. R. Ferguson, of the American Hospital in Britain, skeletal fixation will prove to be of inestimable value in military surgery.

This treatment is not recommended in Colles' fractures because the standard procedure is so satisfactory. Often there is

In simple fractures the skin can receive much better attention and massage can be started immediately. In compound frac-
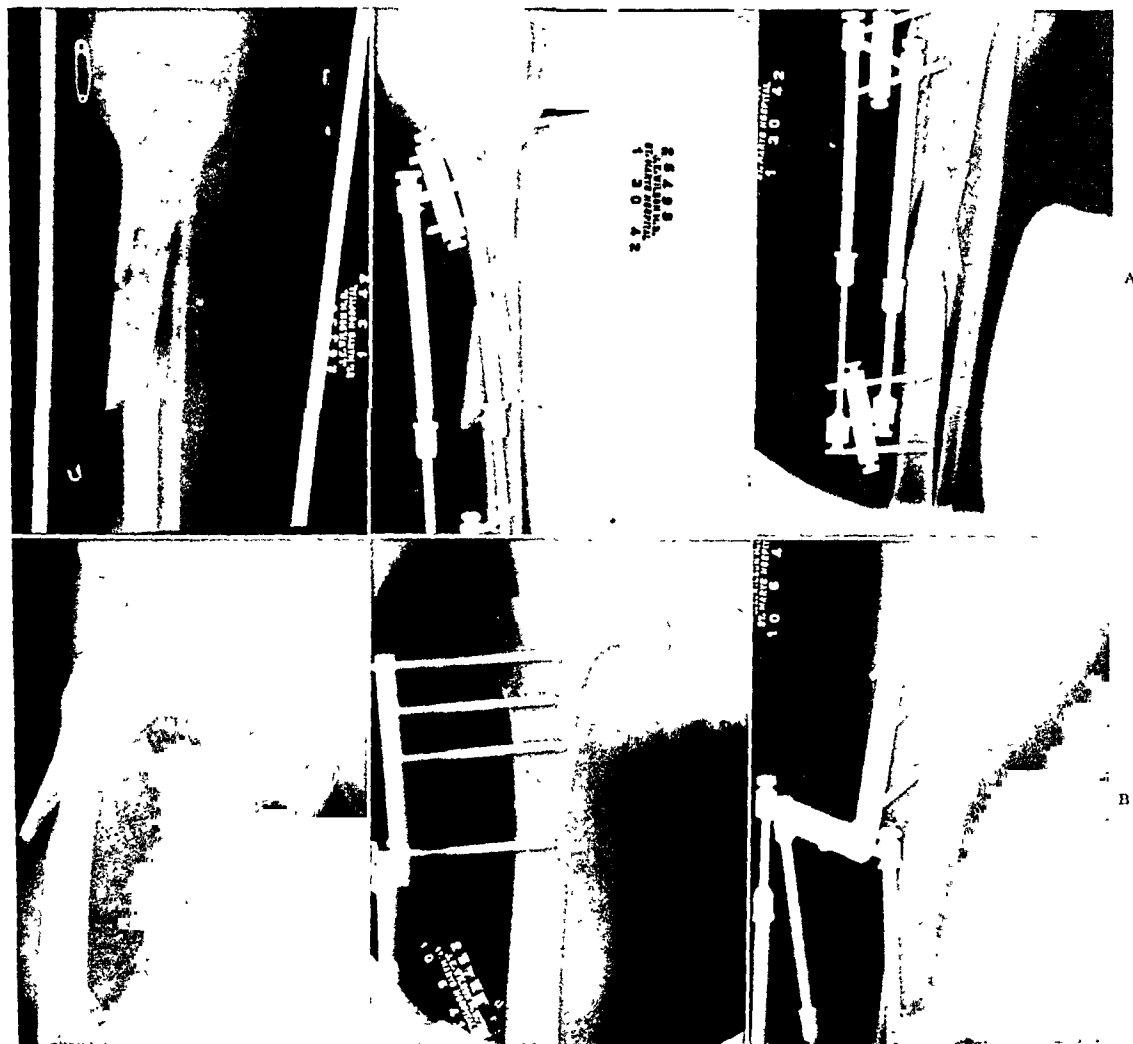
FIG. 7. Fracture of tibia involving the joint. A, anteroposterior and lateral views after reduction. Fixation pin nearest the knee joint is separate from the splint. B, fracture of upper end of femur involving trochanters. Anteroposterior and lateral views after application of splint.

not sufficient room for attachment to the distal fragment. Neither is the treatment advised in simple fractures which are easily reduced with little or no tendency to disapposition or deformity, nor in greenstick fractures.

Foremost among the advantages this treatment has to offer is motion of all joints proximal and distal to the fracture. Pain is almost negligible as soon as the fracture is reduced and the splint locked.

tures the wound can be more adequately cared for and dressed. If desired the Carrel-Dakin treatment can be conveniently carried out. The patient is immediately ambulatory unless more than one limb is involved. Osteoporosis is surprisingly less, due to the use of muscles and joints causing better circulation of blood and lymph. The fact that the pins are used for fixation apparently causes less damage to the bone than when they are used for constant trac-

tion. This conclusion is arrived at from clinical experience and x-ray examination.

Two outstanding cases are here reported, compound, comminuted fracture of tibia and fibula in a twenty-seven-year old white female, in which the pins remained eleven

A   B   C   D
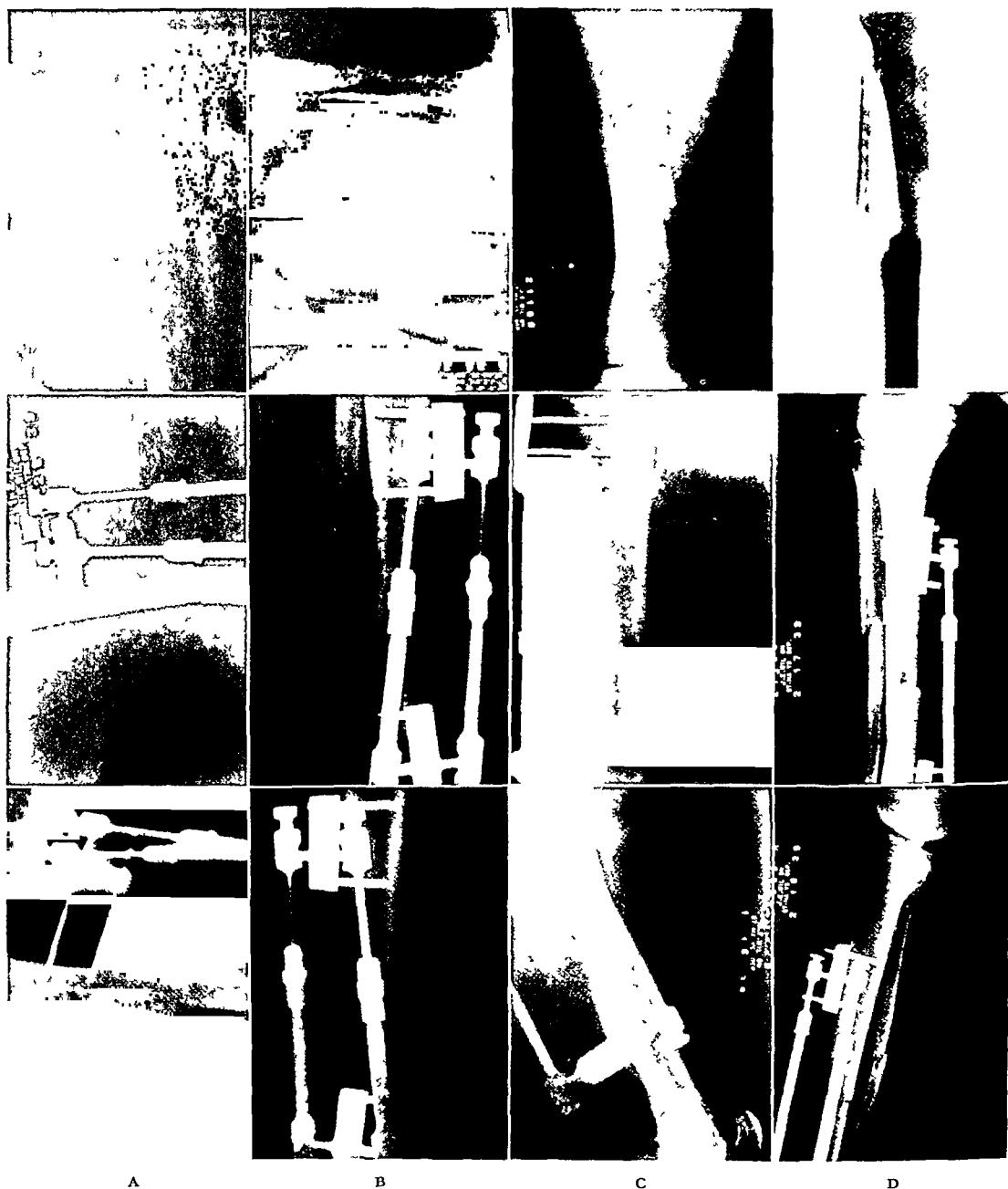
FIG. 8. A, fracture of shaft of the humerus; lateral and anteroposterior views. B, comminuted fracture of tibia and fibula; anteroposterior and lateral views. C, comminuted fracture of the femur; anteroposterior and lateral views. D, transverse fracture of tibia and fibula with loss of bone substance; anteroposterior and lateral views after splint application.

with photographs of x-ray plates and the report by Doctor Firor of the x-ray findings. (Fig. 6.) The first is of bilateral

months. These wounds were grossly infected before the patient was sent to the hospital twenty-four hours after her injury.

In the second case the pins remained *in situ* nine months.

Patients may alter their position in bed

5. Femur—shaft
6. Femur—lower third or fractures about knee
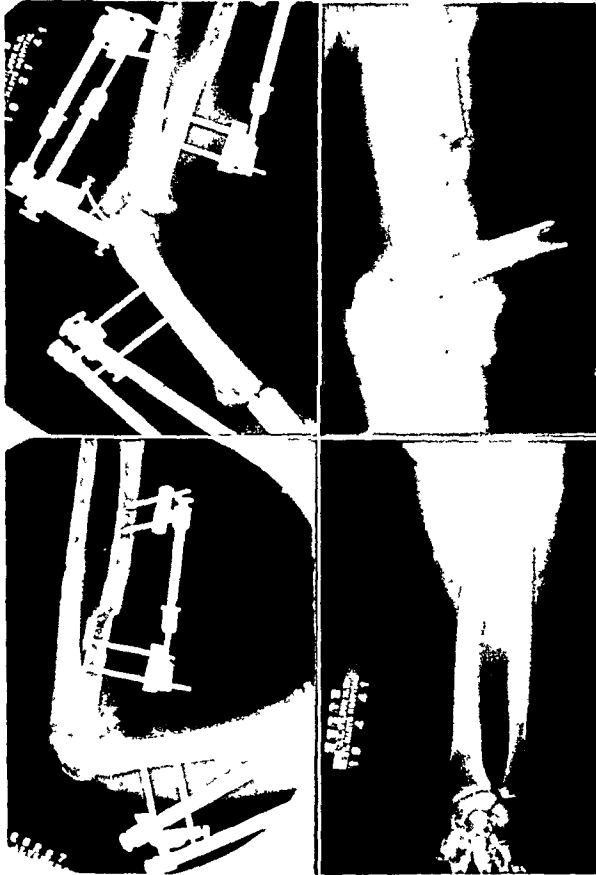
FIG. 9. Car door injury with very extensive damage to soft tissues. Three splints applied: one to humerus, one to radius and one to olecranon.

without help. Consequently, less nursing is required and inspection and mensuration is greatly simplified. The periods of hospitalization and disability are both very materially shortened and ordinary clothing can be worn over the splints. After the first few days it is exceptional to see any swelling of the limb, unless the fracture is compound and there is extensive soft tissue damage.

Splints have been designed for fractures in ten body areas as follows:

1. Humerus—surgical neck
2. Humerus—shaft
3. Humerus—lower third or fractures about elbow
4. Forearm—radius or ulna

7. Tibia—shaft
8. Patella
9. Olecranon
10. Mandible

Since the application of the instrument requires the most rigid aseptic conditions and technic, the procedure lends itself only to hospital and operating room practice and should never be attempted in the office or home. With modern transportation facilities and temporary splinting, any fracture requiring skeletal fixation can be hospitalized.

Points in the technic common to the application of all types of splints are as follows: Careful roentgenographic studies

should disclose the position of the displaced members as well as the type and site of the fracture. From the roentgenogram the type

diameter of the bone it will slip off instead of starting. (Fig. 1.) To prevent soft tissue trauma the bivalved sleeve is placed
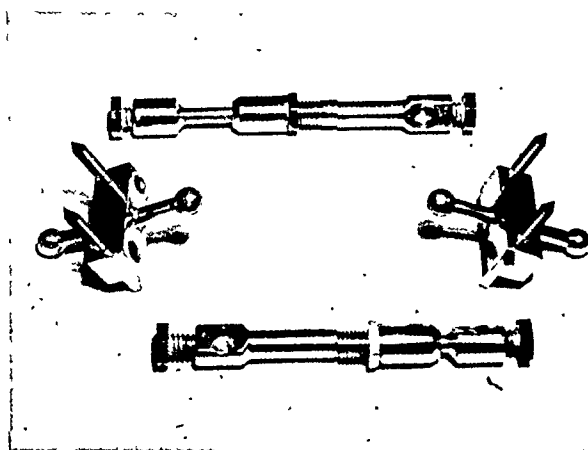
FIG. 10. Disarticulated radius splint with two bars. This is the simplest splint but the principle of all is the same, being two anchoring piers with connecting bars.

of appliance required as well as the approximate length of the bone pins needed can be ascertained.

Anesthesia. The application can be carried out under local anesthesia, but in most instances a choice of intravenous or a short acting general anesthesia is to be preferred. For fractures of the lower limb small doses of a spinal anesthetic agent are preferable.

Preparation. The skin is prepared by shaving, after which it is scrubbed with green soap and water, followed by a sterile bichloride of mercury solution. This is followed by scrubbing with ether, alcohol and then the application of iodine or any of the accepted skin disinfectants.

When using half pins the site for attaching the block to the proximal fragment is then selected. It is important to place the soft tissues at rest, as nearly as possible in their normal position, before the pins are introduced. The block should be well away from the line of fracture. A stab incision 1 cm. long is made through the skin overlying the bone. Through this incision the threaded half pin is introduced down to the bone and after finding the approximate midpoint, the pin is started into the bone. If the pin is placed on the outer part of the

around the pin and worked down to the bone surface and held there while the pin is inserted into the cortex. The sleeve is removed and the pin screwed on across the medullary canal. An increased resistance is noticed when the opposite side of the cortex is reached after which the point is firmly seated in the cortex, by taking two to four additional turns depending on the size of the bone.

The protruding head of the pin is inserted in the proper hole in the block, and the pin and block are used as a guide to find the proper site for insertion of the second pin. This second pin is then introduced through a stab wound in the skin in a similar manner as described above, except that the hole through the block serves as a guide. The technic of insertion of the pins and attaching the block to the distal fragment is a repetition of the above procedure.

The blocks are secured to the inserted pins by tightening the set screws in the sides of the block. A light alcohol dressing is then applied about the pins and blocks.

By manipulation and traction, preferably under the fluroscope, the displacement is corrected. With the fragments held

in place by an assistant, or by the reduction apparatus described below, the bars are secured to the blocks and the friction

When a yoke is applied to one fragment and a block to the other, it is necessary to modify the block. This is done by using a
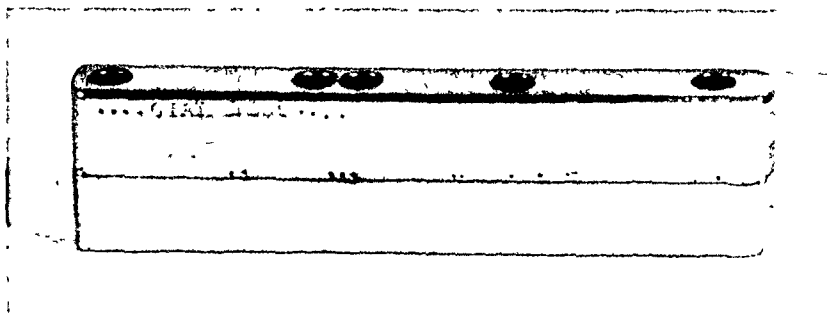
FIG. 11. Bennett block suggested by Dr. George E. Bennett. To be used as a guide for introducing pins. After dressings are applied to protect the wounds, blocks and bars may be applied without the necessity of sterilization.

clamps in the ends of the bars are tightened on the spheres attached to the block. The split sleeve locks in the middle of the bars are then tightened, thus fixing the fractured bone and maintaining position by skeletal fixation.

It is always difficult to manipulate a very muscular limb, even with clamps on the block. For this reason a reduction apparatus has very recently been designed which attaches to the blocks. With this it is possible to get motion in three planes, plus extension. Motion in each plane is obtained by a worm gear which maintains fixation of fragments in any position. The bars are then applied, maintaining any desired position permanently. (Fig. 5.)

In those instances in which it is preferable to use yokes and through-and-through pins, the application is simplified, especially if any one of a number of standard guides is used. As pointed out by Rogers Anderson,[6] it is important that the pins are not placed parallel to each other. After the pins are introduced, both ends of both pins are attached to the yoke and securely locked in the collars. The yokes and pins by their firm attachment to the bone can be used to aid very materially in manipulation and extension. After reduction, fixation is accomplished by application of the bars to the yokes in the above described manner.

crescentic support on which are mounted multiple spheres. The support is attached to the block by a universal friction joint. When the bridging device firmly connects the yoke and the crescentic support, the block and yoke are held in definite relation to each other.

No appreciable amount of slipping has been noticed in any instance in which two or more pins have been securely anchored in each pier. In a few cases of extremely severe injuries, with extensive loss of both bone and soft tissues, pins have been anchored near the joints in a thin spicule not covered by skin. These held about twenty days until they began to show evidence of loosening.

The splint can be removed with very little pain, making the use of an anesthetic unnecessary. First, the hexagonal ends of the connecting bars are grasped to maintain stability. Then the connecting bars are removed, after which a fair estimate of the amount of union can be determined by manipulation of the yokes or blocks. If union is sufficient, the blocks or yokes are disconnected from the pins. At this point the wound and pins should be very carefully cleansed with alcohol and painted with a dependable antiseptic. When through-and-through pins have been used, the end of the pin, which is intended to be drawn through the limb, should be

meticulously cleansed and removed by the ordinary method of removing Steinman pins. After the pins have been removed, an ordinary piston syringe is used to force 70 per cent alcohol through the wound.

The half pins are removed simply by unscrewing them with a tap wrench. When the pins have been started they will turn out with little resistance. After the pins are removed a cannula is passed to the bottom of the tract left by removal of the pin. Seventy per cent alcohol is injected to flush out the tract from the bottom. The skin surfaces of the pin wounds are again cleansed after which dry dressings are applied.

The intention of this article is to show the underlying principles and mechanics of skeletal fixation and demonstrate that the pins hold with a minimum of bone absorption. Case histories have been omitted because they have little if any bearing on the subject.

It should be clearly understood that anyone who attempts to use this method of treating fractures, who is not familiar with the principles of bone and joint surgery, and has not a thorough knowledge of anatomy, is inviting disaster.*

REFERENCES

1. SPEED, KELLOGG. Fractures and Dislocations. Philadelphia, 1916. Lea & Febiger.
2. HACKENBRUCH. Ztschr. f. Arztl., p. 28, 1913; Zentralbl. f. Chir., p. 605, 1913; Deutsche Ztschr. f. Chir., 122: 464.
3. PATTERSON. Am. J. Orth. Surg., p. 649, 1913.
4. GERSTER. Am. J. Surg., 28, No. 31; Am. J. Med. Sc., 146: 157, 1913.
5. CONN, HAROLD R. Internal fixation of fractures. J. Bone & Joint Surg., 13: 261, 1931.
6. ANDERSON, ROGERS. Am. J. Surg., 39: 538, 1938.

COMMINUTED FRACTURES AND FRACTURE DISLOCATIONS OF THE BODY OF THE ASTRAGALUS

OPERATIVE TREATMENT

HARRY C. BLAIR, M.D.

Assistant Clinical Professor of Orthopedic Surgery, University of Oregon Medical School

PORTLAND, OREGON

CRUSHING fractures of the astragalar body, as well as fracture dislocations of the body of this bone, are in practically all instances followed by severe disability. Although most surgeons, according to reports in the literature, are of the opinion that these fractures are best managed by operative measures, there is no unanimity of thought with regard to the type of operative procedure.

My interest in fractures of this type was increased when a sixty-year old man, who had sustained a fracture dislocation of the body of the astragalus, was referred to me for correction of the foot deformity which had followed removal of the fractured astragalar body. The neck and the head of the astragalus had not been disturbed. Because of his age, this patient was not a good subject for remedial surgery. Yet, it was my belief that his partial astragalectomy would ultimately produce a crippling disability. I, therefore, decided to ankylose the tibia to the undisturbed neck of the astragalus by means of a sliding graft from the anterior surface of the tibia. The result was excellent. Some time later I performed a similar operation on a man who had a severely comminuted fracture of the astragalar body. The result in the second case was as good as that in the first.

TECHNIC

The ankle joint is exposed by means of an anterolateral incision, which permits both the removal of the sliding tibial graft used in this operation and the embedding of the graft into the neck of the astragalus. Speed and Boyd,[12] as well as Campbell,[3] who have used the anterolateral incision for ankle fusions as well as for removal of sliding grafts from the anterior surface of the tibia, have already called attention to the wide exposure of the ankle joint which this incision affords. For the technic of the incision, I have drawn freely from the description contained in Campbell's "Operative Orthopedics."[3]

Description of Incision. The incision (Fig. 1A) is started on the anterolateral aspect of the leg, slightly in front of the fibula and about 3 inches above the ankle joint. It is continued distally to about the base of the metatarsal bones, crossing the ankle joint approximately at the juncture of the fibula and the tibia. The fascia as well as the transverse crural and the cruciate crural ligaments are incised down to the periosteum of the tibia and the capsule of the ankle joint. As a rule, both the anterolateral malleolar and the lateral tarsal arteries are also divided by this dissection. Following this, the extensor digitorium brevis muscle is divided in the direction of its fibers, or, if necessary, is detached from the site of its origin. Lastly, the superficial and the deep structures are incised, exposing the lower third of the tibia, the ankle joint, and the neck and body of the astragalus. Throughout the dissection care is taken not to damage any important structures.

Management of Fragmented Astragalar Body. After the ankle joint and the adjoining bones have been widely exposed, the fragments of the fractured astragalar body are taken out. As removal of the fragments progresses, it becomes easier to reach the fragments which lie in the deep portion of the joint. Neither the neck nor the head

of the astragalus are disturbed, since these two parts of the astragalus as well as the articular facets (anterior and medial cal-

A.   B.

C.
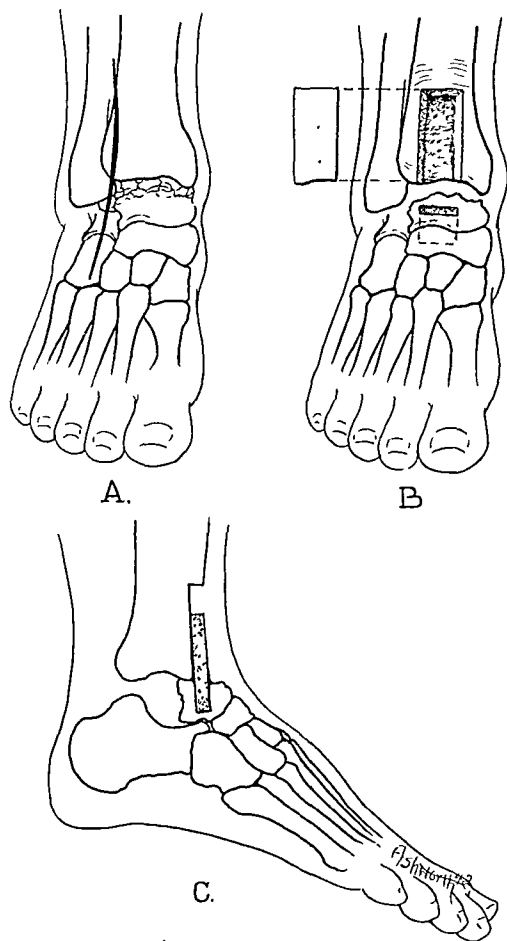
FIG. 1. Schematic drawing of fusion operation for the treatment of comminuted fractures and fracture dislocations of the body of the astragalus. A, anterolateral incision. B, sliding graft from distal, anterior surface of tibia and quadrilateral hole gouged in neck of astragalus. The sliding graft has been removed to permit a better view of the hole in the astragalar neck. C, sliding graft embedded in hole in astragalar neck. Note space left by removal of astragalar body, figures B and C.

caneal) joining them to the os calcis and the astragaloscaphoid joint are ordinarily not damaged.

Preparation and Fixation of the Sliding Graft. When removal of the bony astragalar fragments has been completed, a sliding graft, 1 inch wide and 2 inches long, is cut in the distal, anterior portion of the

tibia. (Fig. 1B.) No cartilage is removed from the articular surface of the tibia except that which is attached to the distal end of the graft.

After the graft has been prepared, a quadrilateral hole is gouged in the neck of the astragalus. (Fig. 1B.) The hole should be about ¾ of an inch deep, and just large enough so that the end of the graft, when it is embedded, will fit snugly.

The foot is then placed in 100 to 105 degrees of equinus, and the previously prepared graft is slipped down and embedded in the hole gouged in the astragalar neck. (Fig. 1C.) The lower end of the graft can be further secured by packing pieces of cancellous bone around it. The upper end of the graft is held in place with sutures taken through the adjacent periosteum and the soft tissues over it.

Closure and Application of Cast. The incision is closed in the usual manner. A gauze dressing is applied over the wound, and sterile sheet wadding is placed over the gauze dressing. A cast is then applied from the groin to the toes, with the knee in extension.

Subsequent Care. Ten to fifteen days after the operation, the cast is taken off; the skin stitches are removed; and a nonpadded walking cast is applied from the knee to the toes. The second cast is equipped with a walking iron, since the patient is encouraged to walk as soon as he is able to bear weight.

CASE REPORTS

CASE I. C. R. W., a white male, age sixty, was admitted to St. Vincent's hospital on December 3, 1937. Ten days prior to admission, he had sustained a fracture dislocation of the body of the right astragalus. (Figs. 2A and B.) The neck and the head of the astragalus, as is usual in these cases, had not been damaged by the injury. Because of the impending necrosis of the soft tissues in the posteromedial region of the ankle, the attending doctor had removed the dislocated astragalar body.

Examination disclosed a severe equinus deformity of the right foot. On the medial side of the ankle, which was extremely swollen, was a

normally healing incision with the stitches still *in situ*. Manipulation of the foot elicited considerable pain. A roentgenogram (Fig. 3) showed that the head and the greater part of

circulatory disturbance, however, persisted for two or three months, and was attributed to injury of the posterior tibial artery at the time the fracture dislocation was sustained.

FIG. 2. A and B, anteroposterior and lateral views of fracture dislocation of the body of the astragalus in Case I. (Films submitted by patient.)

the neck of the astragalus were in their normal position. The body of this bone and a portion of the neck were absent.

On January 24, 1938, approximately seven weeks after the injury, the operation described in this paper was carried out. The tibio-astragaloid joint was exposed by means of the anterolateral incision, and a graft from the anterior surface of the tibia was slipped into the hole gouged in the neck of the astragalus. Pieces of cancellous bone were used for additional packing around the lower end of the graft. The foot was placed in slight equinus to prevent "rocker foot," and a cast was applied from the groin to the toes.

Except for circulatory changes, as evidenced by coldness and duskiness of the skin of the forefoot, the immediate postoperative course was uneventful. Fifteen days after the operation, a nonpadded walking cast with a walking iron was applied, and a few days later the patient was discharged from the hospital. The

Three and a half years after the operation the patient returned for re-examination, stating that he had had no pain during this interval. It was noted that both the foot and the ankle were of normal conformation. There was a small amount of flexion and extension of the ankle, but lateral motion in the distal tarsal joints was almost normal. The functional result was, however, good since the patient was able to run up and down stairs and to climb a ladder without discomfort. The roentgenogram taken at the time of this examination is shown in Figure 4.

CASE II. C. F., a white male, age fifty-four, was admitted to St. Vincent's hospital with a swollen and inflamed left foot on June 29, 1941. He stated that he had fallen from a ladder on June 18, 1941. Roentgenograms showed a severely comminuted fracture of the body of the left astragalus. (Figs. 5A and B.)

The swelling and other local reactions were allowed to subside before operation was

attempted. On July 2, 1941, the ankle joint was exposed through the anterolateral incision. When the body of the astragalus was examined,

the astragalus, according to the procedure described.

Recovery was uncomplicated. A walking cast

FIG. 3. Appearance of ankle joint in Case 1 following initial operation which was performed because of impending necrosis of the soft tissues. Note the undisturbed neck and head of the astragalus and the absence of the astragalar body. (Film submitted by patient.)

FIG. 4. End result in Case 1. This film was taken three and a half years after the performance of the corrective operation described in this paper.

it was observed that the comminution and the displacement were far more severe than the roentgenogram had indicated. There were four

was applied at the end of ten days, but weight bearing was not attempted for at least six weeks following the operation. At the end of four months, however, the patient was walking with only the aid of a leather anklet. Exami-

A   B

FIG. 5. A and B, anteroposterior and lateral views of comminuted fracture of the body of the astragalus in Case II.

or five fragments, and each one was displaced. All of the fragments were removed, and a sliding tibial graft was embedded in the neck of

nation at that time showed practically no swelling, and the patient complained of little pain. A roentgenogram (Fig. 6) revealed that

fusion had already taken place. Further increase in the amount of bone in this area is expected.

DISCUSSION

The diversity of opinion relative to treatment of comminuted fractures and fracture dislocations of the body of the astragalus indicates that no one method has been entirely adequate. Some surgeons believe that dislocated fragments should be replaced when the astragalar body has been fractured.[1,5,10] Others believe that the fragments should be removed.[1] And some of those who favor astragalectomy include with this procedure backward displacement of the foot.[13,14]

Miller and Baker,[9] after their study of the various customary methods of treatment, counseled against astragalectomy, but advised subastragalar arthrodesis when reduction of fragments is not possible. Magnuson[8] was of the opinion that it is often impossible to replace the fragments of a seriously fractured astragalar body, to hold them in place, and subsequently to secure an ankle joint upon which it is comfortable to walk. He maintained that under these conditions it is necessary to arthrodese the ankle, but he stressed the fact that it is "no mean feat of surgery and should not be attempted without previous experience." According to Magnuson, this procedure gives a firm bony union between the astragalus and the tibia, and results in a painless, stable, useful foot with a minimum spring in the arch. There is, however, from 1 to 1.5 inches of shortening. Figure 253 in Magnuson's book on fractures shows an arthrodesis with fusion of the tibia to the os calcis and the neck of the astragalus.

According to Böhler,[1] compression of the body of the astragalus is rare. It was his belief that the body of this bone should, if possible, not be taken out because its removal causes severe and lasting disability. But Wilson[14] took the stand that removal of this portion of the astragalus is necessary since there is usually a great deal of displacement and the circulation is cut

off. He drew attention to a compression fracture of the astragalar body which he called a "check fracture."

FIG. 6. End result in Case 11. This film was taken four months after removal of the fragmented astragalar body and sliding graft fusion of the tibia to the neck of the astragalus. Despite the roentgen appearance, only the middle and the anterior subastragaloid joints remain.

Shands,[11] following an analysis of 109 cases of fractures and dislocations of the tarsus, concluded that astragalectomy may be indicated, but that the results following this operation are not altogether satisfactory. And Hosford,[7] although he had at times been obliged to perform astragalectomy, was also inclined to believe that it does not give an adequately functioning foot. Then again, Campbell[3] in a small series of cases found that astragalectomy had given good results.

Gibson and Inkster[6] maintained that the prognosis in crushing fracture dislocations is poor. In their opinion, partial astragalectomy is unsatisfactory but complete astragalectomy may be satisfactory. Cabot and Binney[2] believed that the prognosis is apt to be poor, and went so far as to say that no operative procedure is likely to improve the condition.

In spite of the arguments in favor of astragalectomy, the fact remains that following astragalectomy the affected ex-

tremity is from 1 to 1.5 inches short. The foot itself is obviously deformed and the fitting of shoes is extremely difficult. Then, too, deformity is liable to be progressive,

displacement is not necessary. In fact, the relationship of the foot to the ankle and the leg remains practically normal and there is no shortening of the extremity.
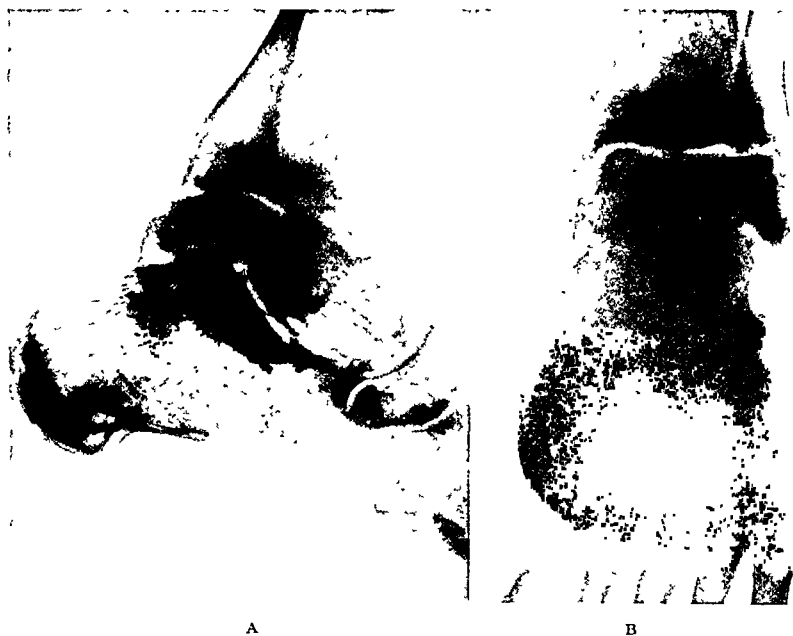
FIG. 7. A and B, end result of a comminuted fracture of the astragalar body which was not managed according to the procedure outlined in this paper. Note the collapse of the astragalar body and nature's effort to fuse the anterior part of the tibia to the astragalus (in A). Note also the traumatic arthritis which subsequently developed (in B). The small amount of motion which remained in this joint was exceedingly painful.

and varus, particularly, is prone to occur. In addition, the adult patient generally continues to have more or less pain, which can be attributed to the fact that weight bearing is not on a normal joint.

Subastragalar arthrodesis and panastragalar arthrodesis, although they have supporters, also do not give entirely satisfactory end results. In comminuted fractures of the body of the astragalus it would seem that both the superior and the inferior joints are involved. Hence, if arthrodesis is to be done, the ankle joint as well as the subastragaloid joints should be fused. Arthrodesis of all these joints should give a painless foot. Yet there would certainly be not only stiffness of the ankle and most of the tarsal joints, but also some shortening.

The fusion operation I have described herein does not change the outward appearance of the foot. Furthermore, backward

An important consideration in this fusion operation is that the weight bearing thrust is placed on normal, undisturbed joint tissue. In astragalectomy, on the other hand, tissue not intended for weight bearing is called upon to carry out this task. The fusion operation now reported should, and actually does, allow flexion and extension of the foot on the leg. The two subastragalar facets and the astragaloscaphoid joint allow a rocking movement of greater or less degree.

Another important consideration in the fusion operation is that there is no tendency for the development of lateral deformity of the foot. It is, therefore, suggested that this procedure might also be considered in those cases in which paralytic calcaneus deformity is present without impairment of the lateral muscle groups.

SUMMARY

1. The customary treatment for comminuted fractures and fracture dislocations of the body of the astragalus has been astragalectomy or tarsal fusion. Results have, however, not been good.

2. An operation of technical simplicity is suggested for the management of these injuries. Briefly, the principal steps are (1) exposure of the tibio-astragalar joint by means of an anterolateral incision; (2) removal of the fragmented astragalar body; and (3) embedding of a sliding graft from the distal, anterior surface of the tibia into the neck of the astragalus.

3. The considerations of special importance in this operation are that the weight bearing thrust is placed on normal, undisturbed joint tissue and that there is no tendency for subsequent lateral deformity of the foot.

4. Experience indicates that the end result of this procedure should be a painless, normal appearing foot and ankle with good stability and function.

REFERENCES

1. Böhler, L. Fractures of the talus (astragalus). Ed. 4, pt. 2, p. 458. In the treatment of fractures (English translation by E. W. Hey Groves). Baltimore, 1935. William Wood & Company.
2. Cabot, Hugh, and Binney, Horace. Fractures of the os calcis and astragalus. *Ann. Surg.*, 45: 51, 1907.
3. Campbell, Willis C. Operative Orthopedics. Pp. 147–148. St. Louis, 1939. C. V. Mosby Company.
4. Davis, Benjamin F. Fracture-dislocation of the astragalus. *Surg. Clin. Chicago*, 3: 1461, 1919.
5. Ely, Leonard W. Old fracture of the tarsus. *Ann. Surg.*, 45: 69, 1907.
6. Gibson, Alexander, and Inkster, Robert G. Fractures of the talus. *Canad. M. A. J.*, 31: 357, 1934.
7. Hosford, J. P. Discussion on fractures in the region of the ankle-joint. *Proc. Roy. Soc. Med.*, 25: 1087, 1932.
8. Magnuson, Paul B. Fractures. Ed. 2, chap. 19, p. 314. Philadelphia, 1936. J. P. Lippincott Company.
9. Miller, Oscar Lee, and Baker, Lenox Dial. Fracture and fracture-dislocation of the astragalus. *South. M. J.*, 32: 125, 1939.
10. Montgomery, Albert H. Fractures through the neck of the astragalus. *Ann. Surg.*, 65: 495, 1917.
11. Shands, A. R., Jr. Fractures and dislocations of the tarsus: an analysis of a series of 109 cases. *South. M. J.*, 24: 1019, 1931.
12. Speed, J. S. and Boyd, H. B. Operative reconstruction of malunited fractures about the ankle joint. *J. Bone & Joint Surg.*, 18: 270, 1936.
13. Wilson, Philip D. Fractures and dislocations of the tarsal bones. *South. M. J.*, 26: 833, 1933.
14. Idem. Fractures and Dislocations of the Tarsal Bones, in Massachusetts General Hospital: Management of Fractures and Dislocations. Chap. 21, p. 544. Philadelphia, 1938. J. B. Lippincott Company.

THE LEVATOR ANI, COCCYGEUS AND PIRIFORMIS MUSCLES*

AGENTS IN THE CAUSATION OF COCCYGODYNIA, SUPERIOR GLUTEAL PAIN AND SCIATIC SYNDROME

THOMAS WILENSKY, M.D.

NEW YORK, NEW YORK

THE difficulties associated with the establishment of an accurate anatomopathologic diagnosis are many in the perplexing and complicated cases which have as their presenting complaint pain in the low-back area. The loose and conflicting usage of terms such as strain, sprain, sacroiliac subluxation, postural fault, congenital anomalies, fascitis, myofascitis, fibrositis, contractures, spastic piriformitis, herniated intervertebral disks, thickened ligamentum flavum, narrowed intervertebral spaces, and sciatica has contributed significantly to the confusion which besets this problem.

Each combination of cause and effect, whether conceived through sound observational pathways or via the exhilarating route of spontaneous inspiration, had its champions and its day of overemphasis.

There can be little satisfaction in conjuring up a composite picture of the female organs sacrificed on the altar of low-back pain. At the present time it is the intervertebral disk that rests uneasily in the warm rays of the spotlight. While recognizing that rupture of a lumbar intervertebral disk with pressure on one or more roots of the cauda equina is a very real and distressing condition, accurately diagnosable in a large percentage of cases and brilliantly cured by skilled surgical intervention, it is conceded that the precisely similar clinical syndrome may be produced by other mechanisms.

Barr, Hampton and Mixter (1937) stated that a clinical differential diagnosis in many cases is not possible. They emphasize the importance of a carefully taken history, thorough physical examination, detailed neurologic study and ordinary roentgenological investigation. Whenever disease of the disk is strongly suspected they advise lumbar puncture, chemical examination of the cerebrospinal fluid for total proteins and in many cases the use of lipiodal or air instillations for more accurate x-ray study of the lumbar spine.

The authors make no mention of the use of routine pelvic and rectal examinations in order to rule out regional disturbances which may be responsible for symptoms similar to those under consideration.

A review of the anatomy of the region displaying the intimate relations existing between sharp unyielding ligaments, actively contracting muscle bundles and relatively huge nerve trunks and blood vessels together with the bones and joints of the pelvis should excite more than a passing interest in these structures from the standpoint of pain arising in and around them.

The sciatic nerve is the longest in the body and subject to baneful influence throughout its entire length. Woltman states that no type of pain is pathognomonic. A given disease may exhibit many kinds of pain which, being subjective, necessitates a most meticulous and cautious interpretation. Moreover remissions of pain are common in the various low-back and sciatic syndrome states, and the frequency of low-back pain as a manifestation of neurosis has been frequently emphasized.

The sacral plexus is formed by the lumbosacral trunk from the fourth and fifth lumbar and by the anterior divisions

of sacral nerves 1-2 and 3. The plexus lies on the anterior surface of the piriformis muscle in 90 per cent of cases and within the muscle in 10 per cent of cases. Morris' Anatomy states that the sciatic trunk may pass above or below the piriformis muscle or the trunk may split and pass around the muscle or the nerve may perforate the muscle. Also there may be a splitting of both nerve and muscle giving rise to any possible arrangement of the four parts. In 10 per cent of cases the piriformis muscle is perforated by the common peroneal nerve. The sacral plexus lies behind the pelvic fascia and the branches of the hypogastric artery. It is also dorsal to intestinal coils with the sigmoid colon in front of the left plexus and the lower part of the ileum in front of the right plexus.

The piriformis is the only muscle which may be said to bridge the sacroiliac joint and it has very intimate anatomical relations with it and also with the sciatic nerve.

In 1928, Yoeman published the first reference to the piriformis muscle as being closely concerned in the production of sciatic pain. He stated that any lesion of the sacroiliac joint can give rise to inflammation in the piriformis muscle and its fascia.

Freiberg and Vinke (1934) published their researches relative to the anatomy and disorders of the piriformis muscle. They discovered the muscle-nerve variations and concluded from clinical studies that sciatic pain may occur when the fibers of the plexus perforate the piriformis and spasm takes place.

Freiberg and Vinke suggested that the relief of sciatic pain by manipulative procedures may have its explanation in the release of adhesions between the piriformis muscle and the nerve sheath rather than from stretching of the nerve trunk which they believe must be a most infrequent result of straight leg raising and other manipulative or limb stretching maneuvers. They suggest, too, that perhaps it is not too radical to consider surgical attack upon the

tendon of the piriformis at its trochanteric attachment in some cases of recalcitrant and incapacitating pain.

In a later article Freiberg (1941) reported upon twelve patients who had been subjected to piriformis muscle interruption. Three of the twelve demonstrated absence or great weakness of the Achilles reflex, a finding considered very significant in cases in which prolapse of an intervertebral disk is suspected. Nevertheless two of these patients (who apparently were not systematically investigated from the disk standpoint) experienced complete relief from pain following operation. The third case was a failure.

Stockman (1920) described fibrositis in great detail. He considered it to be an inflammation of the white fibrous supporting tissue of the muscle. A low-grade serofibrinous exudate was first produced which eventually gave rise to fibrous and tendinous contractures or capsular thickenings.

The Fifth Rheumatism Review (1939) accepted the term, for want of a better, as indicating the syndrome of muscular tenderness, particularly at insertions, and pain on stretching of the part.

In this condition there is characteristic tenderness at the periosteal attachments, and there may be pain and tenderness along the segmental distribution i.e., sciatic syndrome. The clinical reports and pathologic studies by Gratz point to a sustained interest in the musculo-aponeurotic and ligamentous origin of low-back pain.

Heyman believes that the clinical evidence is convincing enough to place fibrositis as one of the three most common forms of rheumatic disease, the other two being atrophic and hypertrophic arthritis. He, therefore, concludes that fibrositis is a provisionally accepted entity causing lowback and sciatic pain which may be relieved by posterior fasciotomy in selected cases not yielding to conservative treatment. His operation is directed toward producing release of tension of the ligaments and fascias and muscles attached to the posterior iliac spine and to the posterior

one-third of the crest of the ilium by subperiosteal stripping laterally, medially and inferiorly, together with division of the sacrospinalis fascia when pain or tenderness is present there.

Heyman reports twenty-six such operations with cures in twenty and partial relief in three. Two of the failures were the result of operations performed for insufficient indications.

Ober's fascia lata section is devised upon a very similar basis. He reports excellent results in carefully selected patients. Ober reported one case in which a ruptured nucleus pulposus had been removed and spinal fusion performed. This patient had persistence of pain which was completely relieved after his particular brand of fasciotomy had been produced. He states that when the patient is suffering greatly fasciotomy can be considered a conservative procedure.

Using the foregoing to indicate the lack of accord which besets this problem, it is our aim to bring to the fore the most important consideration of regional and referred pain resulting from inflammation and spasm in the levator ani, coccygeus and piriformis muscles and their fascias.

In 1937, Thiele reported a series of eighty patients with coccygodynia and/or pain in the supragluteal region and/or pain down the back of the thigh who were treated by massage of the pelvic muscles listed above. He reported 60 per cent cured, 33.7 per cent definitely improved and 6.3 per cent unimproved. Thiele believes that there is a sound anatomic basis for the causation of coccygodynia by spasm of the levator ani and coccygeus muscles and for the production of supragluteal pain and pain down the back of the thigh by spasm of the piriformis muscle.

In thirty-one of thirty-three patients with pain in the supragluteal region or down the back of the thigh he found piriformis muscle spasm. In this general classification of patients, coccygodynia is the more common finding and oftentimes it is so severe that the supragluteal pain or its

radiation down the back of the thigh is elicited only on direct questioning.

Since I was introduced to this syndrome I have searched for, discovered and treated twenty-eight patients. The following eight cases are reported briefly as demonstrating varying points of interest:

CASE REPORTS

CASE I. Mr. L. S., a truck driver, suffered with pain of extreme severity in the right supragluteal region and down the back of the thigh, of sudden onset following a twisting injury sustained when he slipped while carrying a large can of cream. He stated that he had been treated by a traumatic surgeon retained by his company during a period of three weeks. X-rays of his spine had been taken and he had been subjected to various methods of manipulative and physiotherapy. Experiencing no relief he had independently consulted an osteopath with no better fortune. Careful physical examination revealed the one outstanding finding of an extreme degree of tenderness and spasm of the right piriformis muscle. The muscle was massaged thoroughly and he was instructed to apply heat to the area by the use of sitz baths and a heating pad and to return in two days. He failed to return and the writer was left with the impression that the patient had probably returned to the osteopath as the lesser of two evils. Several weeks later I was most agreeably surprised to learn, from the patient's wife that he had not returned because he had been entirely free from pain following the first treatment and that he had not considered it necessary. This patient has been followed for nearly three years and he has never had a recurrence.

CASE II. Mrs. W. T., age twenty-seven, had been delivered of her first child by radical operative intervention including trial forceps delivery, deep episiotomy and finally a laboriously executed version and extraction five months before appearing at my office. She had consulted two gynecologists seeking relief from pain situated deeply in the vagina and a sense

of weight referred to the rectum. She complained also of a dull constant sacral backache which she stated "seemed to empty into the rectum." Both gynecologists carried out endocervical cauterizations without effecting any change in the grade and character of the complaints. My diagnosis was bilateral low-grade inflammatory infiltration and spasm of the levator ani and coccygeus muscles. Four massages carried out at three day intervals succeeded in completely relieving the complaints. Interestingly enough, this patient was delivered easily of her second child eight months ago. She is now under my care again for treatment of levator ani-coccygeus muscle spasm and associated pain and ache, right-sided meralgia paresthetica of moderate severity and external hemmorrhoids grade 1, and skin tags. Massages and physiotherapy in the form of heat served to provide complete relief.

CASE III. Mrs. S. S., twenty-four years of age, when first seen was about three months along in her first pregnancy and complained of severe backache. She stated that at seventeen, while dancing professionally, she had fallen and had injured her back, following which she had never been free from backache. She believed that the pregnancy had aggravated the backache which was situated in both supragluteal and sacroiliac regions radiating to the tip of the coccyx. Examination revealed the positive findings of marked tenderness and spasm, bilaterally, of the coccygeus and levator ani muscles. It was not possible to palpate accurately the piriformis muscles in this patient. Five massages served to provide her with the first lasting relief she had experienced since her fall seven years previously.

CASE IV. Mrs. C. B., twenty-six years of age, suffered with coccygodynia which developed during the course of her second pregnancy. This condition was completely relieved by two digital massages of the levator ani and coccygeus muscles. It is instructive to observe that this patient subsequently returned complaining of "floppy" sensations in her abdomen and pelvis. A remarkably mobile retroverted uterus was satisfactorily "anchored" in the anteverted position by means of an intravaginal pessary. There was no pain or backache associated with this uterine displacement.

CASE V. Mr. H. M., thirty-four years of age, a farmer and factory employee, had bilateral superior gluteal pain which radiated down the thighs and was greatly aggravated by riding the tractor. X-ray films were negative. Bilateral massage of the piriformis, levator ani and coccygeus muscles and associated ligaments was carried out for eight treatments with marked improvement.

CASE VI. Mrs. F. J., forty-six years of age, a picker in onion fields, suffered with an extreme degree of coccygodynia associated with lumbosacral pain radiating down the right thigh to the knee. In this patient the involved muscles and ligaments were board-like to palpation and were extremely sensitive for the first two or three treatments. She received twelve massages and ten graduated hypodermically administered doses of a milk extract foreign protein. She was discharged much improved and was instructed to return for reexamination and possibly for an additional course of treatments.

CASE VII. Mrs. C. H., twenty years of age, had recently been through a very traumatizing forceps delivery of a nine pound first baby. A markedly painful classical coccygodynia was totally relieved by six massages directed bilaterally to the levator ani and coccygeus muscles and fascias.

CASE VIII. Mrs. O. H., thirty-six years of age, had undergone cholecystectomy and appendectomy in 1931. In an automobile accident in 1940 she sustained a fracture of the right humerus and pelvis. Recovery was protracted but complete. In June, 1941, supravaginal hysterectomy was performed for the removal of a rapidly growing fibroid uterus associated with severe pelvic pain and backache. Recovery was uneventful but for persistence of severe backache which radiated down the right thigh posteriorly and from the lumbodorsal and right sacroiliac region to the right femoral head and greater trochanter. Examination revealed marked spastic piriformitis and levator ani and coccygeus muscle spasm especially on the right side. Eight treatments produced gratifying subjective improvement although the muscles remained abnormally firm to palpation.

EXAMINATION

Levator ani and coccygeus muscles are easily felt with the patient in the lithotomy or Sims position. Spasm and tenderness are discoverable by lateroposterior digital palpation. The piriformis muscle is much more

difficult to grasp with the finger-tip. I have discovered that it is most easily reached when the patient is in the exaggerated lithotomy position. Then the muscle can be definitely located just beyond the sacrospinous ligament.

The muscle can be felt to belly out when the extended thigh is externally rotated. In heavy, thick set patients particularly in whom the anal canal is long, narrow and unyielding I have been unable to reach the piriformis. However, when it can be reached, which is most often the case, its texture and sensitivity can be helpfully compared with its fellow on the opposite side.

Freiberg described a sign of piriformis spasm which consists of limitation of motion in inward rotation of the fully extended thigh.

The digital elicitation of pain and tenderness is a more positive finding than might be suspected. The patients complain bitterly of the pain and tenderness whereas the unaffected side may be actively manipulated without the production of more than passing discomfort.

In the dissecting room it has been demonstrated that the sciatic nerve can be pinched between the lower border of the piriformis muscle and the upper border of the sacrospinous ligament. Similarly the superior gluteal nerve can be pinched between the lower border of the gluteus medius muscle and the upper border of the piriformis.

TREATMENT

Massage was originally used to discover whether or not the digitally diagnosed muscle spasm could be relieved by this simple method. In the event that the patient does not show a definite degree of subjective improvement after the first four to six massages over a period of approximately two weeks, orthopedic or other consultation should be sought.

Thiele's series had an average of eleven treatments over an average period of eleven weeks.

Wilson, discussing Thiele's paper, pointed out the interesting finding that eight out of twenty-two patients in his series complained of pain in the lower portion of the abdomen. In every instance this pain followed the course of the iliohypogastric and ilioinguinal nerves and could be exaggerated by pressure on the piriformis muscle near its origin on the affected side. Six of this group of eight cases were relieved by massage of the piriformis muscle alone and the other two were cured by a combination of anal operation and massage.

COMMENT

Obviously there remains a great deal to be done and said concerning this general problem of low-back pain, coccygodynia and sciatic syndrome. It has not been the intention of the writer, in this discussion, to give the impression that the described conditions and the indicated nerve-musculofascial relationships constitute definite clinical entities for which satisfactory treatment can be invariably delivered.

On the contrary it is the express purpose of this paper to emphasize the loose linkage of cause and effect as indicated for these states and to point out that so much of the evidence may be classed as circumstantial.

Nevertheless it is the author's conviction that the muscles and related structures herein considered can and do serve as the direct or indirect causative agents in various cases of low-back and sciatic syndrome pain. Therefore, all such states have not been adequately studied unless this consideration has been explored.

REFERENCES

1. BARR, J. S., HAMPTON, A. O. and MIXTER, W. J. Pain low in the back and "siatica" due to lesions of the intervertebral disks. J. A. M. A., 109: 1765, 1937.
2. CRAIG, W. M. and WALSH, M. N. Neuroanatomic and physiologic aspects and significance of sciatica. J. Bone & Joint Surg., 23: 417, 1941.
3. ELY, L. W. Coccygodynia. J. A. M. A., 44: 968, 1910.
4. FREIBERG, A. H. Sciatic pain—its clinical significance. Ohio State M. J., 30: 21, 1934.
5. FREIBERG, A. H. Sciatic pain and its relief by operation on muscle and fascia. Arch. Surg., 34: 337, 1937.

6. Freiberg, A. H. Fascial elements in associated low-back and sciatic pain. *J. Bone & Joint Surg.*, 23: 478, 1941.

7. Freiberg, A. H. and Vinke, T. H. Sciatica and the sacroiliac joint. *J. Bone & Joint Surg.*, 16: 176, 1934.

8. Gratz, C. M. Fascial adhesions in pain low in back and arthritis. *J. A. M. A.*, 111: 1813, 1938.

9. Gratz, C. M. and Melker, L. H. Role of fascia in myosynovitis and adhesions. *Surg., Gynec. & Obst.*, 69: 627, 1939.

10. Harman, J. B. Fibrositis and pain. *Ann. Rheumat. Dis.*, 2: 101, 1940.

11. Heyman, C. H. Relief of low-back pain and sciatica by release of fascia and muscle. *J. Bone & Joint Surg.*, 23: 474, 1941.

12. Ober, F. R. Fasciotomy. *J. Bone & Joint Surg.*, 23: 471, 1941.

13. Steel, W. A. The relief of chronic backache and sciatica by minor surgical measures. *New England J. Med.*, 219: 474, 1938.

14. Stockman, R. Treatment of sciatica due to fibrositis. *Ann. Rheumat. Dis.*, 2: 77, 1940.

15. Thiele, G. H. Coccygodynia and pain in the superior gluteal region. *J. A. M. A.*, 109: 1271, 1937.

16. Yeoman, W. Relation of arthritis of the sacroiliac joint to sciatica with analysis of 100 cases. *Lancet*, 2: 1119, 1928.

In a condition such as osteo-arthritis of the hip, manipulation often has been performed with the patient under the influence of anesthesia, and sometimes considerable relief has been obtained thereby. It has been suggested that the administration of an anesthetic agent may play a part in the production of such relief.

From "Clinical Anesthesia" by John S. Lundy (W. B. Saunders Company).

CESAREAN SECTION

EVALUATION OF TYPES OF SECTION AND THEIR INDICATIONS

EDWARD A. SCHUMANN, M.D.

PHILADELPHIA, PENNSYLVANIA

SEVERAL extensive recent surveys have again focussed the mind of the writer upon that topic of perennial controversy and comment, the cesarean section.

A significant debate upon this entire subject followed the presentation of a paper by Dr. Edward L. King before the American Gynecological society in 1940. Dr. King's paper was entitled "A Comparison of Two Cesarean Section Surveys Carried on in the City of New Orleans."

The first survey ran from 1921 to 1926 and included an analysis of 300 cesarean sections; these constituted 0.48 per cent of all deliveries and 1.8 per cent of hospital deliveries. The second survey ran from 1927 to 1936, during which time 1,108 sections had been performed; these represented 1.19 per cent of all deliveries and 2.05 per cent of patients who were hospitalized. In the first series the maternal mortality rate was 16.1 per cent, while in the second it had dropped to 5.9 per cent.

In the first group 3 per cent of all patients operated upon died of sepsis or peritonitis, while in the second group 2.25 per cent plus, perished from the same cause.

In the first group 58.3 per cent were treated by classical sections with the probability that many of the eighty-four sections not described as to type were of the classical variety.

In the second group the classical sections had fallen to 49 per cent.

The febrile morbidity in the two series was 63.5 and 61.3 per cent, respectively. This brief summary of Dr. King's statistics is quoted because it is the basis for the comments which follow.

Dr. King's conclusions, among others, were that the average rate of 1.19 per cent cesarean sections in New Orleans was too high a proportion, although there were possibly not enough sections performed during the period covered by the first survey; that the superiority of the low cervical operation is clearly shown and that the indications are more clearly set forth in the records of the past few years and on the whole seem to be more judiciously placed in the second series.

In the discussion which followed the above presentation, several phases of the subject were considered which seem to merit further comment: (1) Is the increase in the relative number of cesarean sections, now the general practise throughout the country, justified or not? (2) The mortality rates of the operation have decreased appreciably during the past twenty years. Why? (3) Is the low cervical section so greatly preferable to the classical type that the latter should be pronounced obsolete as advocated by Dr. Lee in the discussion of King's paper.

With regard to the first proposition, the writer believes strongly that with the steady improvement of technic the cesarean section should be utilized whenever a difficult labor with its inherent dangers to mother and child is anticipated.

The surgical approach in general has been revolutionized during the past two decades. Procedures formerly invoked only upon life saving indications are now routine in the management of lesions causing moderate illness or discomfort. The great development of gallbladder, intestinal, thoracic and indeed every variety of surgical therapeusis, has come about as a result of the increased safety and the decreased suffering in operative procedures.

Obstetric surgery is bound to follow this general trend, and when, as Adair says, "any maternal mortality in excess of 1 per

after labor has begun, and that the danger of cesarean to both patients varies almost in direct ratio to the length of time inter-

FIG. 1. The uterus has been sutured to the abdominal wall and the formation of the peritoneal flap is begun.
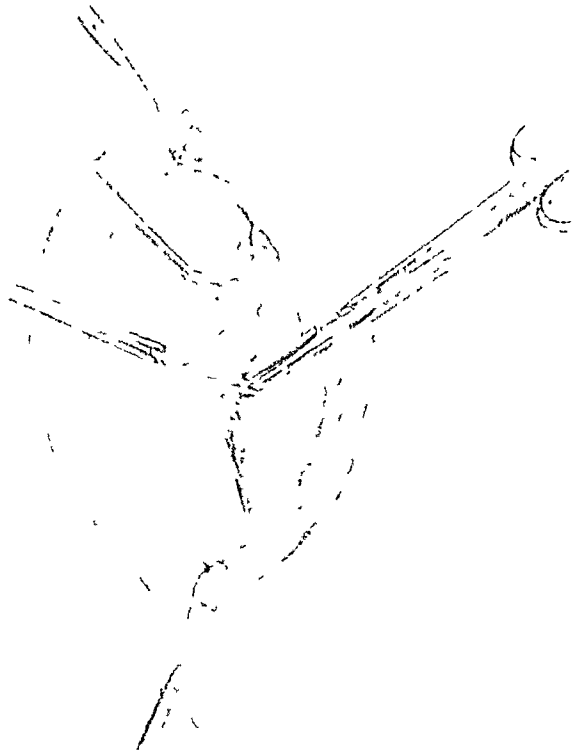
FIG. 2. The peritoneal flap being developed.

cent and any fetal mortality in excess of 2 per cent is too high," it is apparent that section is a safe operation. Danforth surveyed 343 sections done by his own group with a mortality of 0.88 per cent. When it is realized that these figures represent all types of cases, from purely elective to grave emergencies, it is true that abdominal hysterotomy is indeed a safe procedure.

However, a needed advance is the increase in ratio between planned, elective section and those performed as a last resort when all other means of delivery have failed.

The acceptance by the physician of the fact that a stormy labor can be foretold in the great majority of patients long before the event, or at worst, a very few hours

vening between the onset of labor and the performance of the operation, will still further reduce both morbidity and mortality.

On the other hand, there are still too many sections done on infected, exhausted women, when the chance of infant survival is small and the danger to the mother very great. Until some form of truly extraperitoneal section is developed and widely taught (and the ordinary low cervical section is not an extraperitoneal procedure in any sense) embryotomy offers a considerably better prognosis for the mother although the infant is inevitably lost.

In view of the above statements it is my considered opinion that the indications for the truly elective section will continue to be expanded, whereas performance of the section of emergency, especially insofar as it relates to infected patients, will

be more and more curtailed, at least until further knowledge makes the use of extraperitoneal procedures more nearly universal.

transfusion, increase in precision methods of diagnosis, and other details have combined to make surgery in general grow much safer year by year.

FIG 3 The underlying uterine wall is being incised.
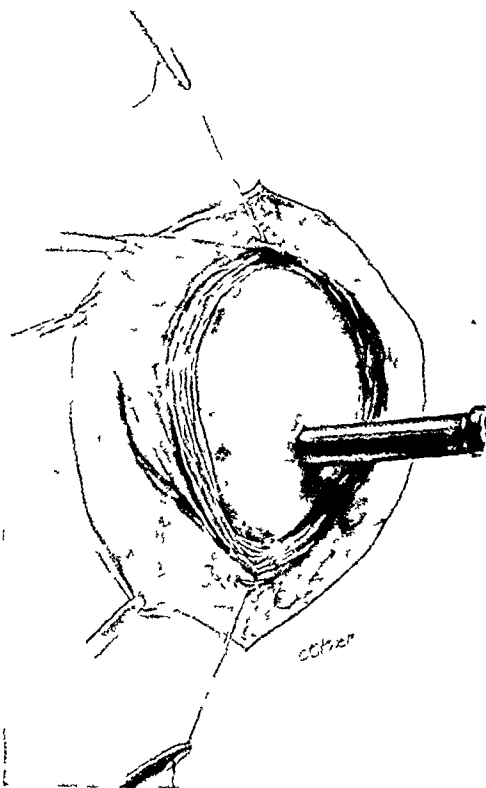
FIG. 4 The membranes bulge through the incision and the liquor amni is being drained off.

Concerning the second question: as to the causes of the manifestly lowered mortality of cesarean sections during the past twenty years or more, one must again turn to the record of surgical achievement as a whole. Certainly a review of the results in cholecystectomy, thyroidectomy, thoracic surgery, compound fractures, indeed any division of operative medicine will reveal an improvement parallel to or even more striking than that disclosed in the reports upon cesarean section.

This advance is due to several factors: Surgeons are better trained than formerly and today, even in the smaller communities, there may be found young men developed in carefully supervised residencies, whose skill and fundamental knowledge is of the highest grade. Surgical technic, anesthesia, the wide use of blood

Insofar as cesarean section is concerned, the same things are true. Kamperman, of Detroit, in the discussion so frequently alluded to in this paper said "In analyzing the statistics, it was interesting to see when the improvement had come. The low cesarean section which had in the meantime become much more popular, seemed to have been partly responsible for a lower maternal mortality. But in my own mind I am not convinced that that was a great factor. We have become more conscious of the danger of cesarean section in the presence of infection and that consciousness developed just about the time when we began to substitute the low cervical section for the classical type. It would seem that knowing when not to operate was an important factor in the improve-

ment. More important still seemed to be the fact that eclampsia was no longer considered an indication for cesarean sections

too loudly as a complete answer to the perils of cesarean section.

The one great place for the low section
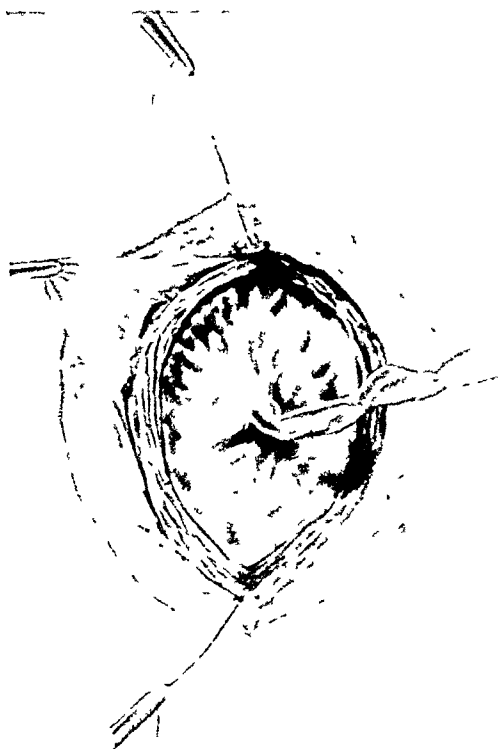
Fig. 5. The baby has been delivered; the placenta presents in the wound.
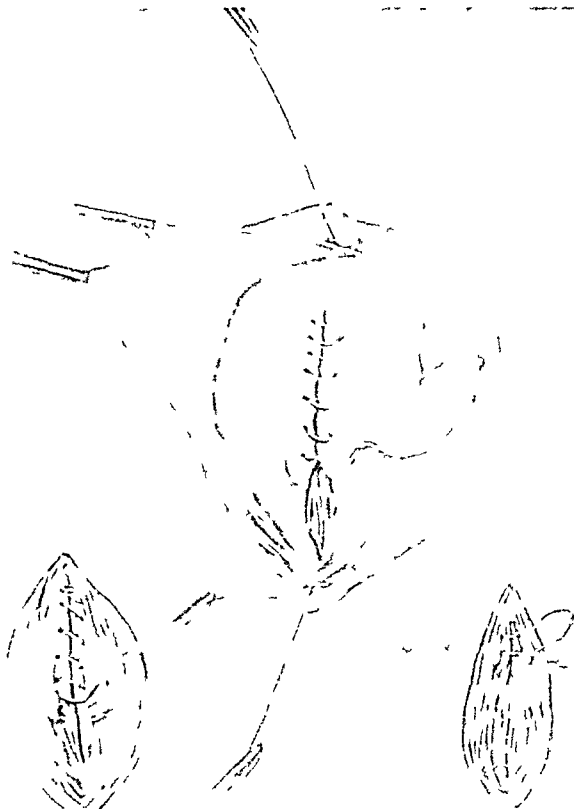
Fig. 6. The uterine muscle is closed.

and I think this change was the chief cause for the improvement in maternal mortality."

All in all, it appears clear that the low section, although it plays some part in the better mortality records, is by no means the overwhelming influence as is so often stated.

The third subject offered for debate is the question as to whether the low cervical section is so far preferable to any type of corporeal hysterotomy that the latter should be pronounced obsolete. The writer has no fault to find with the low section, esteeming it to be an operation intelligently devised and of great value to meet certain well defined indications. He does believe, however, that this procedure has been invested with a certain glamor and hailed

would seem to be in the patient with a failed test of labor, when the membranes have been ruptured for some time and when the lower uterine segment is well thinned out and distended by the pressure of the presenting part. Under such conditions the woman may be potentially infected, and it is reasonable to believe that should suppuration ensue, drainage under the bladder and along the parametrium offers a better chance of localization in an accessible area than occurs if the infected uterine wound lie in the body of the uterus.

It has been claimed that there is less tendency to peritoneal adhesion following section, but if the so-called classical operation be performed with a covering flap of uterine serosa, as will be described, the danger of postoperative adhesion is quite unimportant.

There are claims made for the operation, with which I frankly disagree, a notable one being that the bleeding is less. After
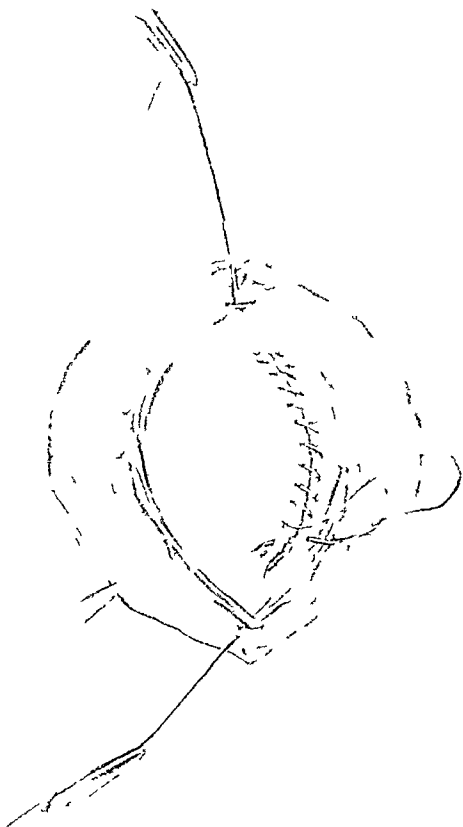
FIG 7 The peritoneal flap completely covers the uterine incision and is being sutured in place

observing many operators in their clinics, one gets the impression that there is practically no difference in the bleeding during a well done section whether it be of the corporeal or cervical type. Indeed, I have seen violent and most alarming hemorrhage occur in the clinics of some of the leading proponents of this operation in America.

It is also commonly said that the low flap operation is quite as simple as the classical procedure, which does not appear to be the fact. It is true that for the trained surgeon the operation offers no difficulties but emphatically it is not as easy and is far more time consuming than the corporeal incision. Furthermore, the tissues

about the urinary bladder are more sensitive and for him who advocates the routine use of local anesthesia in hysterotomy, the time required for successful infiltration is far greater in the low type.

Concerning subsequent rupture of the scar there is but little to choose between the two operative sites. Certainly the lower uterine segment is the seat of most spontaneous ruptures of the uterus and as the low operation is more and more often performed so there will be increasing numbers of scar ruptures reported.

The great point seems to be the increased safety of the cervical section in all cases, as compared to the older form. Three sets of statistics have been adduced which seem to present a fair estimate as to the matter of morbidity.

F. H. Falls of the University of Illinois reported 200 cesarean sections, every other one done by the classical, alternating with the low cervical. The operations were performed in the same room, Falls opening and closing the skin personally. The same nurses and interns were in attendance and the patients received the same pre- and postoperative care. The result of the study was that there is practically no difference between the two operations.

In analyzing the amount of postoperative distress, $\frac{1}{12}$ gr. more morphine was used to keep the patients comfortable who had classical sections than was required for those having low cervicals. Falls says "we feel that the evidence in this series for the low cervical is not very strong and in the hands of the same operators about the same results will be obtained."

Carl Henry Davis found in checking the temperature morbidity that in his classical sections there was a slightly lower incidence of morbidity (51 per cent) than in the low cervical cases (60 per cent). In truly infected patients it is my opinion that no type of hysterotomy in which the peritoneal cavity is invaded is permissible short of hysterectomy. Here vaginal delivery by embryotomy, a truly extraperitoneal operation as the Latzko or the Waters pro-

cedure, sometimes the Gottschalt-Portes exteriorization or removal of the uterus are the only sound methods of attack.

The most recent survey of this subject is that of Ryder* who studied all of the sections performed at the Doctors Hospital, New York, from the opening of the institution in 1930 to the same period 1940. There were 330 sections performed, 218 or 66 per cent being classical and 100 or 30 per cent low flap. The remainder were extraperitoneal or some other variety of operation.

Ryder's conclusions from his study of the statistics were that low flap sections were followed by a higher puerperal morbidity than were the classical in those performed before labor, in electives, in those performed after labor had begun and in all sections considered together, in a survey of all the hysterotomies performed in ten years by many different operators at the Doctors Hospital.

The mortality rate was low, there being four deaths in the 330 operations or 1.2 per cent and the mortalities were twice as high following low flap operations than when the classical variety was employed. Ryder adds, however, that the total number of maternal deaths was so low that conclusions taken from them cannot be very convincing. And also that when the causes of these deaths are studied, a candid opinion must be that the type of section had little to do with the results.

But after all, how often does one meet the neglected, infected type patient? In one urban area (Philadelphia) I have collected the sections done in most of the hospitals during 1940 (to December 1st) and have been surprised to learn that of 580 sections, twenty-four or under 4.2 per cent could be described as being of the neglected dystocia variety. Speaking then for one large city (and I make no comment upon rural areas or other communities, about which I am unacquainted) the num-

ber of these neglected cases has reached a gratifyingly low number.

If it be true, as is so often affirmed, that the low section offers much greater protection against the spreading of an already existing infection than does the corporeal type, it is also true that only one cesarean patient in twenty presents such infection and hence the alleged protection is uncalled for in the remaining 95 per cent.

In this connection it is notable that in certain clinics the test of labor seemed unnecessarily prolonged. Records of active labor of from twelve to thirty hours in the hospital and closely observed with rupture of the membranes of from two to eighteen hours before hysterotomy were noted, and these in women who were known to have pelvic contraction and who had been carefully studied in prenatal dystocia clinics.

It seems that truly elective section long before the point of potential infection and exhaustion of the patient would be a wiser procedure in skilled hands than this excessive dependence upon natural forces which have already shown their incapacity.

From the standpoint of comparative statistics, the cesarean section is unique among surgical operations, especially if an evaluation be attempted of the comparative safety of different types of operation. If one were to make a similar survey of let us say, cholecystectomy, it would be found that the technic in general and the experience and training of the operator would be on much the same level, since no one attempts the operation who has not the necessary skill and the requisite operating facilities.

Cesarean section, however, is a procedure so technically simple that it is performed by almost anyone who practices obstetrics. It is a common thing to hear men who limit themselves to obstetrics announce that they do no abdominal surgery whatever except cesarean sections. This is also true of other physicians who do not even claim especial training in this branch of medicine. The only way in which a definite conclusion may be reached as to

* Ryder, George H. A comparison of the classical and lower segment cesarean section. *Am. J. Obst. & Gynec.*, 41: 1029, 1941.

the value of various technics, is to conduct a series of operations of the different types in the same institution, with the same operators and the same anesthetic, pre- and postoperative management. Then and only then will the true status of corporeal, low segment, and other varieties of hysterotomy be definitely established.

This has been done in part by Falls and by Davis, whose results have been quoted above.

From the foregoing arguments certain opinions of the writer may be summarized as to the place of various types of cesarean section to meet differing indications.

The corporeal or low fundal section is indicated in all elective cases, including operations done for placenta previa and abruptio placentae. This because the method is the simplest, quickest and lends itself most readily to infiltration analgesia. It is just as safe under these circumstances as any other procedure.

The low cervical section is indicated in women who have undergone a test of labor, in whom the lower uterine segment is well distended by the presenting part and who may or may not be potentially infected.

Neither of these operations is wise when definite infection is present, since neither protects the peritoneal cavity against spill. Under such circumstances vaginal delivery by whatever means is available, truly extraperitoneal section, exteriorization or hysterectomy are the only justifiable procedures.

For a number of years I have followed a somewhat specialized technic for the elective section, with satisfactory results for both mother and child. Elective sections are almost all done under local anesthesia, with recourse to inhalation anesthesia in but 1 per cent of the cases.

There have been 204 of these hysterotomies with the loss of no mother and three babies.

There have been no known ruptures of the scar, and in twenty-four of the patients repeat sections have been performed, no adhesions or weakness of the scar being disclosed in any of them.

It must be clearly understood that the operation here advocated is a purely elective procedure, done before the onset of labor or very early in the first stage, the indications being generally cephalopelvic disproportion, toxemia, placenta previa or systemic disease of the mother of a nature in which hysterotomy is deemed the most conservative form of delivery.

In sections of emergency, either the low form or much preferably the Waters technic is advocated. It follows then that the mortality rate here given is not that of section as a whole, but applies only to the low corporeal incision done under local anesthesia as a purely elective procedure.

The technic of the operation about to be described requires for its success meticulous observance of a number of details, insignificant in themselves but all contributing to a smooth convalescence.

The operating team must be trained for silence as noise in the operating room, the skittering of buckets across a tiled floor, clatter of instruments and conversation all tend to awaken and frighten the patient, who has been calmly sleeping before. The obstetrician should always discuss the details of the delivery with the woman, either during the prenatal period or shortly before the operation is due. If the patient be informed that, while she may be cognizant of her operation, any pain will probably be comparable with that occurring during the first hour of labor and that she may rest assured that, should the pain become unbearable the anesthetist is always at hand prepared to administer nitrous oxide or cyclopropane if necessary, she will approach her ordeal with a definite degree of confidence in her knowledge of what is to come.

After proper preparation the patient is brought to the anesthesia room and placed upon the operating table with a pillow under the lumbar spine for greater comfort and, when she is completely arranged, morphine sulfate, gr. ⅙ and

scopalimine hydrobromide, $\frac{1}{150}$ are administered hypodermically. The room is darkened and a nurse sits with the woman until she falls asleep, which usually occurs within fifteen minutes. Thirty minutes after the hypodermic the woman, her eyes lightly covered, is wheeled into the operating room where all is in readiness.

Skin preparation consists of lightly painting the abdomen with iodine and alcohol, after draping the midline is infiltrated with $\frac{1}{2}$ per cent novocaine solution down to the peritoneum. The amount used rarely exceeds 25 cc., 40 cc. being a maximum. A midline incision from the umbilicus downward about 14 cm. is then made, the peritoneum infiltrated and incised. It is most unusual to have the patient complain during the abdominal incision. A No. 2 chromic catgut suture is then introduced at the upper angle of the incision, passing through fascia, muscle and peritoneum then passing into the uterine muscle and out on the opposite side. This suture is tightly tied, the ends held by a hemostat. A similar suture is placed at the lower angle of the incision just at the point of attachment of the vesical fold of the peritoneum. By this means the uterus is anchored to the abdominal incision, and the abdominal walls closely approximate the sides of the uterus. Intestines and omentum are not seen and there is little or no spill of blood and amniotic fluid. Packing is not used. A semilunar incision through the uterine serosa is then made, beginning and ending at the upper and lower sutures and with its convexity toward the operator. A peritoneal flap is very easily turned back by grasping the middle of the incision with an Allis forceps and with a few snips of scissors or simply blunt dissection the flap is turned back until the uterine muscle immediately underlying it is exposed. The uterine wall is then carefully incised for a distance of 2 or 3 cm., until a pouch of membrane bulges through the opening. A gauze pad is laid about the bulging membrane and with trocar and suction apparatus the liquor amnii is drained away.

The field being dry, the uterine incision is lengthened, usually from the upper to the lower suture, either by cutting with scissors or, as is preferable, by tearing the fibers apart with the fingers. It is remarkable to note the approximation of the uterine walls upon the completion of the operation when the uterus has been opened by tearing the fibers. This is because the uterine muscle fibers are not severed but merely displaced and pulled apart and tend to readjust themselves as soon as uterine contractions begin. One cc. of pituitrin is given at the beginning of the uterine incision and the baby is withdrawn by breech extraction. Hemorrhage from the uterine wall is ordinarily negligible but if sinuses bleed freely they may be clamped with T hemostats covered with rubber tubing. The usual time for the separation of the placenta is permitted and this organ generally bulges up into the uterine wound in from five to ten minutes after the extraction of the baby. Should this not be the case, the placenta may be separated in the ordinary manner. The empty uterus is then closed by two layers of interrupted or continuous catgut sutures, depending upon the predeliction of the surgeon; and finally the peritoneal flap, which now extends far beyond its original area, is utilized to cover the suture line in the uterus, being attached by a continuous suture of No. 00 catgut. In order to close the abdomen without permitting intestines or omentum to protrude into the wound, the lower suture attaching the uterus to the abdominal wall is cut first and the parietal peritoneum closed by a continuous suture until the upper attachment is reached. This is then cut and the closure of the peritoneum and the rest of the abdominal wall completed. Should it be desired to sterilize the woman, the lower suture is cut, the uterus gently rotated upon the upper suture until the tube appears and when this structure has been anesthetized by further infiltration, any desired procedure for sterilization may easily be completed.

It has been said earlier in this communication that the writer has no fault to find with the low flap cesarean section but he is convinced that in elective operations it offers no especial advantage over corporeal section and that in frankly infected patients it gives little if any more protection against a spreading peritonitis than does the higher incision, because the peritoneal cavity is equally invaded in both instances. Its greatest field of usefulness lies in its employment in the patient who has undergone several hours of labor under good supervision, but who may be in danger of what is sometimes termed potential infection.

The following is an example of the curious thinking which has led to the widespread opinion that the low flap section is safer and preferable to the low fundal type:

In a special article in the Year Book of Obstetrics and Gynecology for 1940, the late Dr. J. B. De Lee, probably the most enthusiastic proponent of the low section in America, made the following statements. He quoted the statistics of the Chicago Lying-In Hospital 1914 to 1936 in which there were 168 classical sections with eleven deaths (6 per cent) and 1,875 laparotrachelotomies with eighteen deaths (0.96 per cent) and made this comment: "All of the advantages grow out of the position of the opening in the uterus. The lower part of the abdomen stands operation and infection better than any other part; the cervix is an infection buffer," etc.

Here is a categorical statement that the tremendous improvement in mortality of one operation over another lies in the fact that the incision in the favored type lies at a maximum of three inches lower than in the condemned procedure.

Two pages further on in the same article this statement appears:

"In securing such remarkable results with laparotrachelotomy at the Chicago-Lying-In Hospital, credit must be given to two measures. First, a rigid selection of cases. In the last 500 patients there were no suspect infections, and few had had any vaginal examinations. Second, we used local anesthesia in nearly all of the 1,250 cases. Thus we avoided pulmonary complications entirely and, with them, the occasional wound infection and peritonitis which arise by way of the blood stream from the infected lung. Further, we thus reduced the incidence of disturbance in the healing of the abdominal wound. Vomiting after anesthesia puts a strain on the recently placed sutures—some giving way, some tearing the muscle or fascia or fat, leaving dehiscences, hematomas and serous extravasations. General anesthesia also increases the morbidity and the mortality of those diseases for which abdominal delivery may be done, e.g., nephritis, heart disease and tuberculosis. Tympany and stomach disturbances coming from anesthesia are almost unknown after the local infiltration method. Hemorrhage from the wound and from the uterus is less, contraction and involution are much better, and altogether local is a much more physiologic method than general anesthesia."

Obviously, if the second conclusion is true, the first has no weight, and vice versa.

This article is quoted, not to assail the work of Joseph B. De Lee, who was always regarded by the writer with the utmost respect and esteem, but to emphasize the lengths to which enthusiasm and partisanship may lead even in the discussion of dispassionate, scientific problems.

The writer has always been a strong advocate of elective section upon any definite indication but it is an interesting fact that in the clinics under his supervision, in which the average number of deliveries is 1,800 annually, the total number of cesarean sections has never exceeded 2 per cent in any one year and the ten year average is 1.8 per cent, this including neglected clinic cases, and indeed, all sections for whatever reason they have been performed.

It does not follow then, that a belief in the value of this or any other procedure need lead to its injudicious employment. The increasing safety of cesarean sections, whatever type of operation is performed has led to certain abuses which have tended to again discredit the operation, despite the improvement in technic and results.

I refer to the indiscriminate employ-ment of hysterotomy, to satisfy either the desire of the patient to avoid labor or to conserve the time of the obstetrician and the fatigue engendered by attendance upon spontaneous delivery. Although elective cesarean section is one of the safest of operations, it is still far more dangerous to the mother than vaginal delivery and always carries with it certain subsequent risks.

IN the clamp method of removing the puerperal uterus, there are no ligatures or sutures used, and this is an important factor in the surgery of this type of infection as there is no foreign material remaining to prolong the infection.
From "Vaginal Hysterectomy" by James William Kennedy and Archibald Donald Campbell (F. A. Davis Company).

POSTOPERATIVE HERNIA

REPORT OF A CASE OF REPAIR WITH FASCIAL SUTURES

WILLIAM BERRY MARBURY, M.D.

Clinical Professor of Surgery, George Washington University School of Medicine

WASHINGTON, D. C.

A POSTOPERATIVE hernia may occur in any part of the abdominal wall which has been opened surgically. It undoubtedly has its inception during the healing of the incisional wound. Of the twenty-one cases of postoperative hernia which were admitted to Emergency Hospital during the period from 1935 to 1940, inclusive, the time of the appearance of the hernia was known in thirteen cases. Three of the thirteen patients felt a mass in the wound during convalescence from the primary operation. Three others noticed the appearance of the hernia during the first year following operation. Thus in 46.1 per cent of the cases the hernia was known to have developed within the first postoperative year. Branch[1] reported that in a series of 300 cases of incisional hernia diagnosed at the Peter Brent Brigham Hospital, 139 (46.3 per cent) of the patients had noticed the presence of a mass in the area of the wound during the first ninety days following operation.

The factors which contribute to the development of a postoperative hernia are of four kinds: (1) The presence in the patient of obesity or of poorly nourished tissues at the time of the primary operation; (2) increased intra-abdominal pressure during the postoperative period; (3) infection and drainage of the incisional wound; and (4) certain types of operations.

Obesity is accompanied by an increase of fat in the omentum. An excessive amount of omental fat makes the primary abdominal operation more difficult; and it also, by increasing the contents of the abdomen, subjects the suture line to added tension. Among 100 patients with incisional hernia whose primary operation had been performed at the Peter Brent Brigham Hospital, Branch[1] found that forty-eight were so obese at the time of their primary operation that it was difficult to make a satisfactory closure. In thirteen of these cases there was no factor other than the obesity to explain the occurrence of the hernia. Smith and Masson[2] have reported that among the eighty-four cases of ventral hernia repaired with sutures of fascia lata at the Mayo Clinic during the years 1933 to 1936, inclusive, twenty-five patients weighed more than 200 pounds. Schlesinger[3] is of the opinion that disturbances of the healing process are more often caused by the sutured tissues being in a state of poor nutrition than by any other factor. It is generally agreed that a blood dycrasia, such as the anemias, diabetes, syphilis, nephritis, or carcinoma, constitutes a predisposing condition to the breakdown of the operative wound.

Increased pressure within the abdomen following operation is associated in some cases with the development of incisional hernia. When the wound is in the upper part of the abdomen, pressure and tension are induced by the patient's coughing, vomiting or hiccoughing. Pressure in the lower portion of the abdomen is induced by distention. Bancroft[4] divides postoperative hernias into two groups: those occurring below the umbilicus and those occurring above the umbilicus, those below being caused largely by infection and distention, those above largely by the forcible excursion of the ribs pulling upon the transversalis fascia during the act of coughing or vomiting. Among the 100 cases studied by Branch,[1] there were forty-three patients who had abdominal distention,

persisting for from twenty-four to forty-eight hours or longer, following their primary operation, and twenty-eight who had a postoperative cough, lasting for several days. Branch believes that distention or a postoperative cough causes an increase in the intra-abdominal pressure, which not infrequently breaks the sutures or causes them to tear through the tissues, thus allowing the viscus to protrude between the remaining sutures and further weaken the incisional line.

<div style="text-align:center">

TABLE I

PRIMARY OPERATION IN TWENTY-ONE CASES
OF POSTOPERATIVE HERNIA

</div>

Operation	No. of Cases
Appendectomy	13
Appendix ruptured in 6 cases, necessitating drainage	
Patient obese in 4 cases	
Cholecystectomy	1
Wound infected; patient obese	
Cholecystectomy and gastroenterostomy	1
Drainage	
Perforated peptic ulcer	1
Drainage	
Salpingo-oophorectomy	1
Wound infected; patient obese	
Prostatectomy	1
Laparotomy	1
Abdominal injury	1
Not stated	1

Infection of the operative wound and prolonged drainage are also important etiological factors of incisional hernia. Branch[1] reported that forty-nine of the 100 patients in his series showed evidence of infection; in thirty-three the incision had been drained and later became infected, and in the remaining sixteen the wound was infected extending down to, and in some cases through, the fascia. In fourteen additional cases the operative wound had been drained although there was no evidence of infection. Among the twenty-one cases of postoperative hernia admitted to Emergency Hospital, infection was a factor in eight. The wound was drained in two additional cases. Infection or drainage was, therefore, a factor in 47.6 per cent of the series. In thirteen cases of the series, the primary operation was performed because of appendicitis. In six the appendix was

ruptured, necessitating drainage. It is noteworthy that four of the thirteen patients were described as being obese; thus infection, drainage, or obesity was a factor in the development of the postoperative hernia in 77 per cent of the appendicial cases. (Table I.) Miscellaneous data concerning the age, sex, and race of the patients in the series from Emergency Hospital are presented in Table II.

Whether a particular type of operation favors the formation of an incisional hernia

<div style="text-align:center">

TABLE II

INCIDENCE OF AGE, SEX, AND COLOR IN TWENTY-ONE
CASES OF POSTOPERATIVE HERNIA

</div>

Age	
20 to 40 years	7
40 to 60 years	9
60 to 80 years	5
Sex	
Male	12
Female	9
Color	
White	18
Colored	3

is debatable. Answers to a questionnaire which McCauliff[5] sent to a number of surgeons showed that 75 per cent of the ruptured wounds which they reported were associated with a midrectus incision and that most of the remaining 25 per cent followed either a transrectus or a midline incision, nearly all occurring above the umbilicus. None of the surgeons recorded the rupture of a muscle-sliding or muscle-retracting incision. Among the 300 cases of incisional hernia admitted to Peter Brent Brigham Hospital, 45 per cent occurred in the lower midline, 17 per cent in the upper right rectus, 16 per cent in the lower right rectus, 15 per cent at McBurney's point following a muscle-splitting incision, and the remaining 7 per cent at scattered points in the abdominal wall. Branch[1] considered that the ratio of 215 females to 85 males in this series is due to the apparent susceptibility of the wounds in the lower midline to rupture and the frequency of this incision in females.

The repair of an incisional hernia is successfully accomplished in the majority of cases by the use of one of the established

methods of herniotomy, such as the over-lapping closure devised by Mayo for the repair of umbilical hernia. In some cases the fascial layers cannot be made to overlap, because they have retracted too widely to be approximated. The ideal procedure of suturing like tissues is impossible when muscle and fascia are bound together by adhesions and cannot be separated. In approximately one-fourth of the cases, the usual methods are inadequate, owing to the thinness of the surrounding muscle and the wide retraction of the fascia having created an extensive defect. With such a defect in the abdominal wall, when it may be impossible to approximate the muscle-fascia edge, plastic devices have to be resorted to.

Gallie and LeMesurier[6] were the first to devise a technic for using strips of auto-genous fascia lata as living sutures in the repair of large hernial openings. Their method is briefly as follows: The hernial sac and its contents are treated in the usual manner. If the muscle and fascia surround-ing the hernial opening cannot easily be dissected, they are left as a single matted layer. When the margins of the opening can be approximated, the defect is closed by a running suture formed from a quarter-inch strip of fascia lata. A meshwork of fascial sutures are then superimposed over the suture line to re-enforce it. When the margins of the hernial opening cannot be approximated, because of the size of the defect or the tension under which the closure is being made, the edges are drawn together as close as is safe and the remain-ing defect is filled with a woven meshwork of fascial sutures placed closely enough to prevent the underlying tissues from pro-truding between them. In reporting their operation in 1923, the authors emphasized three procedures in the successful use of their method: (1) The fascial sutures must be solidly anchored into unyielding tissue, such as healthy muscle or aponeurosis. (2) The fascial sutures must be employed in sufficient quantity to withstand the antici-pated strain. (3) When the margins of the

hernial opening cannot be drawn together, enough suture material must be used to fill the gap completely so that no crevice remains through which the peritoneum can protrude. The authors recommend that the first layer of fascial sutures be overlaid with other layers, which should be woven into strong tissues well back from the edges of the opening.

In reported series of cases of large inguinal hernias repaired with autogenous fascial sutures, the percentages of recur-rences range from 3.6 per cent, in a series of eighty-two cases reported by McCloskey and Lehman,[7] to 7.4 per cent, in a series of fifty-four cases reported by Gray.[8] There is a similar disparity in the reported results obtained in the repair of large ventral hernias. Shelley[9] reported that in a series of 207 cases of repair of incisional hernia which were followed for nine months or longer at St. Lukes Hospital, New York, there was a recurrence rate of 13.0 per cent in the group of twenty-three repaired with fascial sutures, in contrast to a rate of 18.5 per cent recurrence among the 184 repaired by other methods. In the series of 300 cases of incisional hernia which Branch[1] studied, there were fourteen in which narrow strips of transplanted fascia lata were used as running sutures to bridge a large fascial defect. There were three recurrences, mak-ing the percentage of recurrence (21.4 per cent) slightly higher than in the group as a whole (19.6 per cent). Smith and Masson,[2] in 1940, summarized the results achieved at the Mayo Clinic during the four-year period from 1933 to 1936, inclusive, when eighty-four ventral hernias were repaired with sutures of fascia lata. These eighty-four hernias were the most difficult out of a total of 299 ventral hernias operated upon at the Mayo Clinic during that period. Sixteen of the eighty-four hernias had been repaired one to five times previously. There were five recurrences among the eighty-four cases repaired with fascial sutures (8.1 per cent). An average of forty-three inches of fascia lata was used. In one case 100 inches of the suture material was used. The

authors stated that most of the recurrences were in those cases in which a comparatively small amount of the fascial sutures had been used, and that they are now inclined to use more suture material than they did formerly.

In the following case of postoperative hernia, I adapted the Gallie technic, modifying the operation in several respects, to repair a particularly large hernial opening. Gallie used quarter-inch strips of fascia lata, which he allowed to roll into a cord-like suture. In the operation reported below, strips of fascia lata one inch wide were used and were held in place by silk sutures. As the margins of the hernial opening could not be approximated, there was no tissue to be re-enforced. The bands which were applied across the opening were the only covering over the defect.

CASE REPORT

The patient, a rather large, well built man, thirty-five years of age, was seen on March 25, 1940. He complained of an incisional hernia on the right side of the abdomen. He gave a history of having been operated upon, elsewhere, three times. The first operation was for a ruptured appendix, which required drainage. The second and third operations were for the repair of the hernia. Each closure had been made with catgut. Following each operation the hernia had recurred. The last operation had been done in 1936.

The physical examination revealed nothing important except the condition of the abdomen, where a definite protrusion was most marked when the patient stood or coughed. The weakness in the fascia was about three inches or more in diameter and about five or six inches in length. Practically no fascial border could be felt on the outer side toward the anterosuperior spine. The patient was advised that it would be necessary to take a fascial transplant from his thigh, and that even then there could be no guarantee of a cure. He accepted these conditions and was admitted to the hospital several days before the operation. The size of his abdomen was reduced by diet and catharsis. Both the abdomen and the left thigh were prepared in advance as in "orthopedic cases."

The patient was operated upon April 8, 1940, under avertin and gas. The two existing scars were excised and the skin and superficial fascia were dissected back to expose the borders of the combined muscle-fascia planes. The weak area of peritoneum and scar tissue was large enough to admit an adult fist. The peritoneum was opened in one place and dense adhesions were found between coils of small intestines and the undersurface. This opening was closed without further disturbing the abdominal contents. The edges of the incision were dissected and freshened. They were then brought together at the corners and sutured with fine black silk, so as to narrow the opening. A transplanted strip of fascia lata, one inch wide and about eighteen inches long, was woven across the opening and its surrounding area of weakened tissues. The strips were held flat and fastened with silk sutures, as shown in Figure 1. As the dissection was extensive and some oozing was anticipated, a Penrose drain was inserted in each end of the wound.

The day after the operation, the patient's temperature was 104.4°F., his pulse was 110 beats per minute, and he began to cough. A large amount of bloody serous fluid drained from the wound. The leucocyte count was 26,000, with the polymorphonuclears 81 per cent. Dr. B. L. Hardin, Jr., who examined the patient, found dullness in the right middle lobe of both lungs. On the second postoperative day, a roentgenogram of the chest showed infiltration in the region of the hilus, on both sides. Examination of the sputum showed pneumococci, type IX. On the sixth day, there was a semipurulent discharge from the wound. When one or two stitches were removed, a great quantity of this same fluid poured out. The outlook for a permanent cure of the hernia seemed very dubious.

The patient left the hospital on the twenty-second postoperative day. The drainage, although diminished, was still considerable. The wound was dressed in the office every other day, and later once a week, for two months. On several occasions, silk sutures were removed by hooking them with a crochet needle. No fascia was ever seen or removed. The patient has been examined at intervals and there has never been any evidence found of a weakening of the sutured area or of a recurrence of the hernia.

The satisfactory result in this case brings up two controversially interesting points: (1) The advantages of using living fascia in the repair of hernias; and (2) the use of nonabsorbable sutures in infected wounds. A discussion of the advantages of using living fascia as sutures in the repair of hernia may well be prefaced by a statement of the conditions under which its use is justifiable. It is indicated, I believe, only where there is a congenital loss of fascia, as in some cases of inguinal hernia, or where there is an acquired and fairly extensive loss of fascia, as in the case which has just been reported.

The technic of Gallie and LeMesurier[6] was based upon extensive animal experiments which demonstrated the following points: (1) In the healing of incisions made in the fascia and aponeurosis of the experimental animals, the new connective tissue

SILK SUTURE

FIG. 1. Shows cross lacing of the wound with a broad strip of fascia without tension. The strips were approximated closer than shown in the drawing.

over the line of suture was continuous with the areolar membrane normally on the surface of those structures. There was no evidence of any inflammatory reaction in the cells of the fascia or aponeurosis themselves and no indication that they contributed to the production of the scar tissue. Histologically, the union between the cut edges of the fascia and of the aponeurosis appeared to be weaker than the surrounding tissues. When specimens were examined after the lapse of months, it was almost always found that in those specimens in which the edges of the incision had been sutured under tension, or in which the

line of sutures had subsequently been subjected to strain, the edges of the wound had separated, owing to the stretching of the scar. (2) When fascial transplants were sutured to fascia, it was found that the fibers of the fascia did not proliferate during the process of healing. The transplant remained exactly as it had been at operation. It was held in position only by a thin areolar membrane derived from the areolar fibers of the neighboring tissues. When the normal activities of the animal produced strain on the line of healing, the edges of the transplant and the edges of the defect separated just as had the edges of the simple incised wounds. (3) When quarter-inch strips of fascia were used experimentally to bridge a gap of half an inch and were later recovered, it was found that even after two years the fascial sutures remained practically unchanged and that their tensile strength remained unimpaired. No stretching or contraction had occurred. On the basis of their experiments, Gallie and LeMesurier concluded that fascial sutures are superior to patch transplants in the repair of large hernial openings because the living suture depends for its strength not on scar tissue but on the mechanical grip with which it holds the edges of the wound. When used clinically, the autogenous fascial suture does not appear to slough off to any extent in the presence of infection. It also does not seem to stretch and thin out under tension to the degree that scar tissue does.

It has been shown repeatedly that the incidence of hernia is higher in infected wounds than it is in wounds which heal per primam. It is also fairly well established that infection occurs more frequently in wounds sutured with catgut than in those closed with silk. Elkin[10] found that in a series of 1,787 surgical cases the incidence of infection was 2.1 per cent in wounds sutured with silk and 9.4 in wounds closed with catgut. In a series of potentially infected cases he found that definite infection developed in 7.9 per cent of the wounds

sutured with silk, compared to 21.4 per cent in which catgut was used.

The exact cause for the more frequent occurrence of hernia following the use of an absorbable suture than of a nonabsorbable suture is not altogether plain. However, it has been shown by Howes and Harvey[11] and others that catgut is absorbed at different intervals in different individuals and under different conditions. In the presence of infection, the absorption is frequently accelerated. Fibroplasia progresses more slowly when catgut is used than when silk is employed; hence healing is delayed. The complete absorption of catgut requires considerable phagocytosis and tissue reaction. This process also delays healing. One has only to look at a wound in which silk alone has been used and compared it to one in which catgut has been employed to see the difference in the healing process. In the former there is very little sign of inflammation, as evidenced by heat, redness, and swelling, whereas in the latter all three are present.

Before leaving the question of sutures, I should like to quote from Ochsner.[12] In 1939, he stated: "I began using silk in persons who were debilitated, such as patients with carcinoma. I now use silk much more extensively. I feel that silk is particularly indicated in cases of infection. Ordinarily one would think of not using silk in the presence of an infection, but I believe that in cases in which healing is delayed and in which rupture is likely to occur its use is especially indicated. If one does use silk, let me reiterate the points which Professor Halsted emphasized many years ago. One must use fine silk; only interrupted sutures are to be used and the knots cut short; and only small bits of the tissue are caught in the stitch. In spite of the teaching of Halstead and Whipple concerning the proper use of silk, most surgeons when beginning to use silk use it incorrectly and therefore obtain undesirable results. If one will use interrupted silk sutures, it is surprising how many frankly contaminated wounds and infected wounds will heal with-

out discharge. It is continuous silk in the presence of infection which is particularly dangerous." In a more recent communication, Meade and Ochsner[13] proposed cotton as a suture material which is pliable, readily sterilized, nonirritating, and, when used as recommended, sufficiently strong for most surgical procedures. Their proposal was based upon experiments which demonstrated the relative loss of tensile strength, amount of tissue reaction, time of wound healing, and extent of the bacteriology associated with sutures of catgut, silk, linen and cotton. They reported having used cotton sutures in ninety-one surgical cases, in eighty-eight of which primary healing took place.

There are certain precautionary measures which may be carried out in cases of abdominal surgery to lessen the possibility of an incisional hernia developing or recurring. In the preoperative preparation of the patient, excess weight should be reduced; the undernourished patient should be given proper food; colds and other infectious processes should be cleared up, if possible; and the general health of the patient should be improved. Following operation, the patient should remain at rest in bed for an adequate period. If he is inclined to be obese, he should not be permitted to gain weight. The food intake by mouth should be limited. To prevent abdominal distention or vomiting, a Wangensteen suction tube, inserted through the patient's nose, should be employed for two or three days after the operation. Singleton and Blocker,[14] who reviewed 9,000 consecutive cases of abdominal surgery while studying the problem of wound disruption and postoperative hernia, were convinced that gastric suction would prevent postoperative vomiting and distention if the measure were effectively and routinely used during and after operation in all cases of abdominal surgery. Patients usually strain while becoming accustomed to the presence of a suction tube. To protect the wound against the effects of undue abdominal strain, a Skultus bandage, or binder, should be applied

before the tube is placed in position. As prolonged drainage appears to be a predisposing factor in the development of hernia, drainage should be instituted only when it is definitely indicated, and the tubes should be removed as soon as practicable.

SUMMARY

1. The predisposing factors of postoperative hernia have been discussed. Obesity, abdominal distention, coughing, and vomiting, by subjecting the line of suture to increased tension, may cause a breakdown of the incisional wound; infection and prolonged drainage may weaken and disrupt the wound, thus permitting the formation of a hernia.

2. A review of twenty-one cases of postoperative hernia admitted to Emergency Hospital shows that infection or drainage was a factor in 47.6 per cent of the series. Infection, drainage, or obesity was a factor in 77 per cent of the thirteen cases in which the primary operation was an appendectomy.

3. The Gallie technic of using living sutures of fascia lata has been examined. The operative results in representative published series of hernial repair by the use of fascial sutures have been reviewed.

4. A case has been reported in which a modification of the Gallie technic was used in the repair of a large hernial opening.

5. Emphasis has been placed upon the superiority of nonabsorbable sutures in infected wounds and the advantages of using fascial sutures when the edges of the wound are widely retracted.

6. Precautionary measures to guard against the development of hernia have been considered.

REFERENCES

1. BRANCH, CHARLES D. Incisional hernia: analysis of three hundred cases. New England J. Med., 211: 949–952, 1934.
2. SMITH, CALEB H. and MASSON, JAMES C. Results of the repair of ventral hernias with sutures of fascia lata. Review of eighty-five hernias. Surgery, 7: 204–211, 1940.
3. SCHLESINGER, ARTHUR. Die Operation ubergrosser Bauchnarbenbruche. Zentralbl. f. Chir., 59: 1151–1155, 1932.

4. Bancroft, Frederic W. Hernia. *Pennsylvania M. J.*, 40: 63–70, 1936.

5. McCauliff, Guy T. Rupture of wounds following abdominal operations. *J. Iowa M. Soc.*, 23: 347–351, 1933.

6. Gallie, William E. and LeMesurier, A. B. The use of living sutures in operative surgery. *Canad. M. A. J.*, 11: 504–513, 1921; Living sutures in the treatment of hernia. Ibid, 13: 469–480, 1923.

7. McCloskey, John F. and Lehman, James A. Living fascial sutures in the repair of large inguinal herniae. *Ann. Surg.*, 111: 610–615, 1940.

8. Gray, W. The operative treatment of inguinal hernia. Results obtained in adult males by the fascial suture technique. *Brit. M. J.*, 1: 568, 1940.

9. Shelley, Harold J. Ventral hernias. A study of 550 hernias and 458 repairs. *South. Surg.*, 9: 617–656, 1940.

10. Elkin, Daniel C. Wound infection: a comparison of silk and catgut sutures. *Ann. Surg.*, 112: 280–283, 1940.

11. Howes, E. L. and Harvey, S. C. Tissue response to catgut absorption, silk and wound healing, correlation to tensile strength. *Internat. J. Med. & Surg.*, 43: 225–228, 1930.

12. Ochsner, Alton. Discussion: The problem of disruption of abdominal wounds and post-operative hernia. *J. A. M. A.*, 112: 126–127, 1939.

13. Meade, W. H. and Ochsner, Alton. The relative value of catgut, silk, linen, and cotton as suture materials. *Surgery*, 7: 485–514, 1940.

14. Singleton, A. O. and Blocker, T. G., Jr. The problem of disruption of abdominal wounds and postoperative hernia. A review of 9,000 consecutive abdominal incisions. *J. A. M. A.*, 112: 122–126, 1939.

In Pott's fracture, the ankle joint appears widened. The foot is everted. Great swelling appears promptly. Point tenderness is felt over the internal malleolus and over the fibula just above the malleolus.
From "A Manual of the Treatment of Fractures" by John A. Caldwell (Charles C. Thomas).

ADDITIONAL EXPERIENCES WITH SPOOL COTTON AS A SUTURE MATERIAL*

PHIL THOREK, M.D., RALPH GRADMAN, M.D. AND ALFRED GLAESS, M.D.

Associate Surgeon, Cook
County Hospital

Surgical Resident, Cook
County Hospital

Resident Physician, Cook
County Hospital

CHICAGO, ILLINOIS

AN ideal suture material from the surgeon's point of view would be one that induces little tissue reaction, is absolutely sterile and maintains its tensile strength while embedded in tissue. Our experiences in Cook County Hospital during the past two years have convinced us that cotton thread meets the above prerequisites satisfactorily and is superior to catgut, linen or silk. Since cotton is easily sterilized, inexpensive, portable and readily available, it will undoubtedly make an ideal war-time suture material. We have used cotton as the suture of choice in over 400 major and minor surgical procedures with excellent end results.

Cotton is not a new suture material. It was used as early as 500 B.C. by Susruta.[1] Recently, however, it has been popularized by Meade and Ochsner,[2] Meade and Long,[3] Taylor,[4] and Thorek,[5] all of whom report excellent results with cotton thread. To appreciate the advantages of cotton a review of the dynamics of normal wound healing is essential. All that can be expected of a suture is that it will keep the tissues approximated; the process of healing is a natural phenomenon and occurs in spite of the suture used. The approximation of the wound edges is immediately followed by an exudation of serum which tends to glue the tissues together. This constitutes the first phase of wound healing and is followed by the second phase known as fibroplasia. Any factor increasing the exudation phase will accordingly delay the onset of fibroplasia and prolong healing. Catgut heals by "wet healing," whereas most nonabsorbable suture materials are associated with "dry healing." "Wet heal-

ing" merely refers to the prolongation of the serum exudate brought about by the tissue reaction. Cotton, unlike catgut and other suture materials, appears to be least irritating to the tissues and heals by "dry healing." This phenomenon of exudation associated with the various suture materials has recently been clearly demonstrated by Farris[6] who placed catgut, silk and cotton into the anterior chamber of a rabbit's eye. After remaining in situ for fourteen days, the catgut produced a marked opacity of the anterior chamber, practically blinding the animal. Although silk produced a slight reaction it was more irritating then cotton and showed a tendency for its fibers to untwist. Cotton, however, was associated with the least irritation and showed little tendency to untwist and fray at the edges.

Cotton has other qualities which make it superior to silk or linen. Cotton is a vegetable fiber which has a natural tendency to twist. Fraying of cotton does not occur as with linen or silk, thus fascilitating its handling, threading, etc. Meade and Ochsner believe this twisting of fibers associated with cotton prevents the ingrowth of leukocytes and infected granulations into the substance of the suture material. This may be the explanation for the absence of draining sinuses with cotton and this not infrequently occurs with silk, especially when used in infected cases. (Figs. 1, 2 and 3.)

The wound is not healed until fibroplasia is completed. During the ten to twelve days usually required for its completion, the approximation of the wound edges is dependent primarily upon the

tensile strength of the suture material. Meade and Ochsner have shown by animal experimentation that comparison of cotton,

for practical purposes we have used only three, Nos. 100, 50, (quilting) and 24. No. 100 is a very fine cotton thread and must
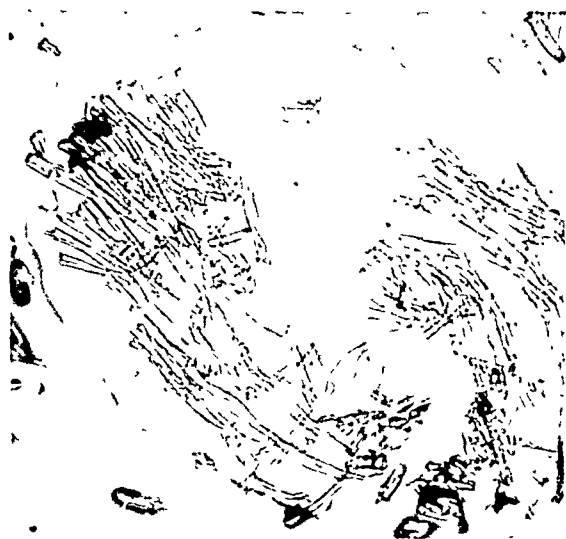
FIG. 1. Quilting cotton magnified 92 times. The fibrils have a natural tendency to twist.

FIG. 2. Pagenstecher linen magnified 92 times. The fibrils appear straight and parallel.

silk, linen, and catgut revealed that cotton was unexcelled in maintaining its tensile strength *in vivo*. After the sutures were embedded in tissues for ten days, cotton maintained its tensile strength at 100 per cent, whereas silk, linen and catgut had a tensile strength loss of 25, 40, and 70 per cent, respectively. The pliability of cotton and its increased coefficient of friction makes handling and knot tying easier and more secure.

Sterilization of cotton thread can be accomplished simply by autoclaving for ten minutes at 15 pounds pressure or boiled for twenty minutes without diminishing its tensile strength. Since cotton contracts when heated it should not be sterilized on a rigid spool. Formerly we wrapped the thread on a flexible rubber tube; the compressibility of the rubber would prevent breaking or loss of tensile strength of the cotton fibrils. Recently, however, we have had satisfactory results by cutting the cotton thread into the desired lengths, attaching them to a cardboard with a paper clip, and autoclaving for ten minutes.

Various sizes of cotton are available, but

be handled gently. It is used for ligation of small vessels and in subcutaneous suturing. Quilting corresponding to No. 50 is the next largest size used and is utilized for closure of peritoneum and skin, seromuscular and purse-string sutures in bowel surgery. No. 24 is the largest size used and with it we repair the fascia in all abdominal incisions and herniorrhaphies, as well as ligation of larger arteries such as the popliteal, superior thyroids, uterine, etc. If for some reason a heavier suture is desirable, we utilize No. 24 doubled.

Our excellent result with cotton can be attributed to the teachings of Halsted,[7] who emphasized that nonabsorbable sutures, (he was primarily referring to silk) should be used interrupted, using only very fine material, and avoiding strangulation of tissue as well as dead space. Although he stressed that absorbable and nonabsorbable sutures should not be used in the same wound, we have found no ill effects when we would occasionally suture peritoneum with continuous catgut whenever as peedy closure was advisable.

Regardless of the suture material used there are other important factors influenc-

ing wound healing, such as nutrition of patient, hypoprotenemia, vitamin c content of blood, local blood supply, presence fare, (appendectomies performed on soldiers in tents without modern methods of insect and fly control) their incidence of infection

FIG. 3 Black silk magnified 92 times The fibrils appear rigid and straight

of infection, skill of surgeon, etc. As pointed out by one of us (P. T.) in a previous communication, tissues requiring approximation under tension do not belong together. Cotton thread will not tolerate rough handling, therefore, by its constant use the surgeon will further the development of a nontraumatic technic. Avoiding tissue strangulation, catching only the vessel and not its adjacent fat, using finer suture material, cutting directly on the knot, and accurate hemostasis will soon result in a greater incidence of primary wound healing.

Other properties of cotton make it the ideal war-time suture. Sparkman and Williams,[8] in a recent communication have demonstrated that cotton has proved its worth during actual United States Army maneuvers in the fall of 1941. Using cotton under conditions simulating actual war-

in 120 cases was 0.83 per cent. Their conclusions were in accord with ours, that cotton can be employed safely in clean, contaminated or infected wounds.

Cotton is readily sterilized, portable, and above all is very inexpensive. Foss[9] has compared the cost of cotton, silk and catgut as used in 647 operations. The average operation required about 25 yards of cotton or silk, and 6 tubes of catgut. The cost of cotton was 1¼ cents, silk 93 cents, and catgut $1.16 per operation. Approximately ninety operations can be performed with cotton for the same cost of one operation using catgut.

Our series at present, over 400 cases, includes such cases as thyroidectomy, mastectomy, gastric resections, gastroenterostomy, Mickulicz procedures for large bowel neoplasm, appendectomy, pelvic surgery, cholecystectomy, common

duct surgery, herniorrhaphies, hydrocele, skin grafts, nerve and tendon repairs, leg amputations, and many minor surgical procedures as repair of fresh lacerations, vein ligations, etc. We have classified all our cases into clean and contaminated wounds; our incidence of infections were less than 4 per cent. Since this includes perforated appendicies and infected diabetic gangrenes, the incidence of infection is exceedingly low. We have intentionally used cotton in grossly contaminated wounds to determine whether cotton like silk would produce a chronic draining sinus. To date no such sinuses have developed. We have especially been impressed with the high incidence of primary union in cases of supracondylar amputations for spreading diabetic gangrene. Prior to the use of cotton, draining sinuses, osteomyelitis of the stump, and necrotic suture lines were not infrequent in our diabetic unit. During the past two years with the adoption of cotton technic these latter complications have indeed become a rarity; of the last twenty leg amputations, two infected wounds resulted. It is interesting to note that in each infected case catgut and silk were the sutures used.

The interrupted suture technic was used in all instances with few exceptions. Continuous No. 00 catgut was used for peritonealization of gallbladder bed, cervical stump following hysterectomy, and for peritoneal closure whenever speed was necessary and for the mucosal suture in intestinal anastomoses. We still believe that in gastrointestinal anastomoses, hemostasis is best accomplished with a continuous mucosal catgut No. 00 suture. We have, however, used quilting cotton as a through-and-through suture on the mucous membrane side in a cholecystogastrostomy and also in a case of Meckel's

diverticulum in which the base of the diverticulum was very wide and had to be sutured by means of a two-layer closure. No ill effects were noticed.

At present we are conducting a series of cases with sulfanilamide and cotton used together. Our results will be reported in a later communication.

CONCLUSION

1. Further experiences with spool cotton as a suture material are reported.

2. Its availability, economy and ease of sterilization makes it an ideal war-time suture.

3. It can be used in grossly contaminated wounds without danger of sinus formation.

4. The routine use of fine cotton will further the development of better surgical technic.

5. Cotton appears to be superior to catgut, silk and linen both experimently and clinically.

REFERENCES

1. Melle, G. J. Early history of the ligature. *South African Med. J.*, 8: 290, 1934.
2. Meade, Wm. H. and Ochsner, Alton. Spool cotton as a suture material. *J. A. M. A.*, 113: 2230–2231, 1939.
3. Meade, W. H. and Long, Carrol H. The use of cotton as a suture material. *J. A. M. A.*, 117: 2140–2143, 1941.
4. Taylor, K. P. A. Single unit cotton thread surgical technique. *Smith Surg.*, 10: 125, 131, 1941.
5. Thorek, P. Experiences with spool cotton as a suture material. *Am. J. Surg.*, 55: 118–120, 1942.
6. Farris, John M. In discussion of Drs. Meade and Longs paper. *J. A. M. A.*, 117, 2141, 2142, 1941.
7. Halsted, W. S. The employment of fine silk in preference to catgut and the advantages of transfixing tissues and vessels in controlling hemorrhage. *J. A. M. A.*, 60: 1119, 1913.
8. Sparkman, Robert S. and Williams, William H. *Surgery*, 11: 698, 1942.
9. Foss, Harold. In discussion on Estes, W. L. Jr. End results in repair of inguinal hernia by a fascia to fascia rectus sheath closure. *Ann. Surg.*, 113: 838, 1941.

GALLSTONE ILEUS*

RECURRENCE IN ONE CASE

B. Hollis Hand, m.d.   AND   William E. Gilmore, m.d.
Resident Surgeon, Hospital   Assistant Resident Surgeon, Hospital
for Women of Maryland   for Women of Maryland

BALTIMORE, MARYLAND

WE believe the three cases presented in this paper are of unusual interest in several respects. All three cases occurred in a 140-bed hospital within a period of approximately two years. The preoperative diagnosis was suggested in the one case, and was made with certainty by the aid of x-ray in the others. One patient had a recurrence of gallstone ileus within the first week of convalescence which was likewise diagnosed and treated successfully surgically.

The recurrence of the obstruction in the first case might have been avoided if a thorough search of the entire small bowel had been made at the first operation. However, the presence of the second stone was unsuspected because at the first exploratory laparotomy the intestinal loop proximal to the stone was collapsed.

INCIDENCE

In a report of 500,000 operative cases by Martin[1] there were only sixteen cases of intestinal obstruction due to gallstones. In 1935, Boreman and Rigler[2] reported eleven cases of gallstone obstruction in 19,474 autopsies at the University of Minnesota Hospital. Judd,[3] in 153 cases of internal biliary fistula operated upon at the Mayo Clinic reported that in 1925 there was only one case of gallstone ileus. However, in 1938, Wakefield, Vickers and Walters[4] reported ten cases of gallstone ileus in the same clinic. In Table I, 12,153 cases of intestinal obstruction are summarized with 208 due to gallstones, i.e., 1.7 per cent. Balch[6] states that women are much more prone to this obstruction than men, in the proportion of 15 to 1, whereas the incidence of gallstones is about 3 to 1 in favor of women.

TABLE I

Total Obstructions	Gallstone Obstructions	Percentage	Authority
3,625	47	1.3	Vick[5] (Symposium on Intestinal Obstruction, 1932)
1,655	28	1.7	Souttar[5] (Symposium on Intestinal Obstruction, 1925)
505	2	0.4	Meyer and Spinack[6] 1934
295	23	7.8	Osler[5] 1900
1,152	41	3.5	Leichtenstern[5]
360	8	2.2	Barnard[5] 1902
669	14	2.1	Barnard[5] 1905
502	10	2.0	Balch[5] (M. G. H. 1898–1932)
3,064	28	0.9	Bennett[7]
179	4	2.2	Powers[18]
147	3	2.	(Hosp. for Women of Md. 1929–1941)
12,153	208	1.7	

DIAGNOSIS

Pybus[9] reported a case in which he could feel a hard object, presumably a gallstone, in the right iliac fossa before operation; this finding is much too rare to be useful in diagnosis. Although the diagnosis may be strongly suspected because of gallbladder disease associated with a bizarre type of intestinal obstruction, a conclusive diagnosis can scarcely be made preoperatively without the aid of x-ray. Rigler[14] mentions definite preoperative diagnosis by x-ray in only twenty-four cases reported up to November 1941. The most helpful sign

leading to diagnosis from x-ray is probably gas or opaque material in the biliary tree. This indicates probable internal biliary

removal of the gallstone, but on examination of the gallbladder two additional stones were found. The gallbladder was

Fig. 1. Case 1. X-ray in routine gallbladder series disclosed (1) gas in biliary tree; (2) dilated small bowel; (3) stone within lumen of small bowel; (4) nonfilling gallbladder. The positive diagnosis was biliary fistula, high intestinal obstruction and gallstone ileus. This stone was proved to consist of cholesterol by chemical analysis.
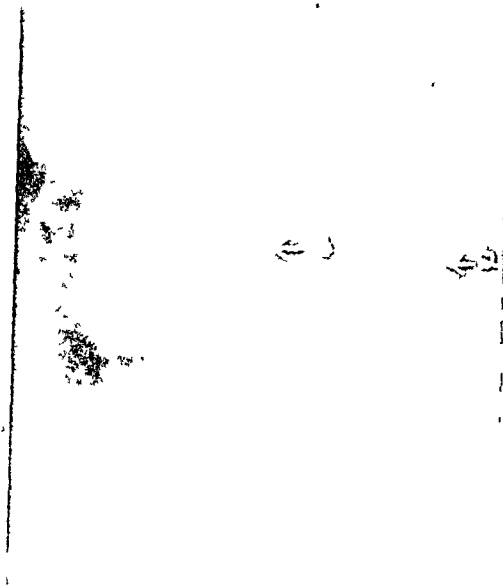
Fig. 2. Case 1. Flat plate of abdomen taken on the eighth postoperative day because of recurrence of obstructive symptoms. The findings were (1) gas in biliary tree; (2) dilated small bowel; (3) stone in lumen of small bowel. The positive diagnosis was recurrence of gallstone ileus on the eighth postoperative day. This stone was proved to consist of cholesterol by chemical analysis.

fistula. A stone in a dilated intestine unfortunately is not often visualized, but when seen is almost conclusive of gallstone ileus. If a biliary fistula can be demonstrated in the presence of intestinal obstruction, the diagnosis is fairly certain. (Fig. 4.) In Figures 1, 2 and 5 the diagnosis is certain.

MULTIPLE STONES

Holz[8] described a case in which a stone as large as a hen's egg was removed from the sigmoid. One large facet faced upward and he presumed another stone to be present higher. The second stone with the opposite facet was found adhering to the duodenum and obstructing it. This was removed along with the gallbladder, and the patient made an uneventful recovery. Pybus[9] reported a case in which the obstruction was relieved by ileotomy and

separated from the duodenum, the opening in the latter closed and the two stones removed from the gallbladder which was simply drained. This patient likewise recovered. Moschowitz[10] presented a case of gallstone ileus relieved by ileotomy, and enteroenterostomy because of postoperative narrowing of the lumen of the ileum. On the twenty-sixth postoperative day a large facetted stone was manually removed from the rectum and the patient was discharged well. Drew[13] reports a case in which the stones were found so impacted in the jejunum as to necessitate two separate openings into the bowel for their removal. These cases and many others[4] indicate the presence of multiple stones without recurrence of serious obstruction. Recurrent ileus is much less common than the findings of multiple stones.

RECURRENT GALLSTONE ILEUS

Downs[10] recalled a case in which he removed one stone in operation only to

FIG. 3 Case I. X-ray taken one month after first ileotomy. It clearly demonstrates biliary tree and indicates the persistence of the internal biliary fistula for at least one month. There is no evidence of obstruction or stone anywhere

have a recurrence of obstruction forty-eight hours later. On re-opening the wound a second stone was found impacted in the gut at the point where the sutures had been inserted. Pybus[9] reported a case of recurrent obstruction four days after removal of a large stone from the terminal ileum. The symptoms were thought to be due to peritonitis and no further operation was performed. At autopsy, "General Peritonitis was found due to a small perforation of the ileum above a stone impacted about 5 feet above the ileo-cecal valve and about two feet above the incision for the removal of the first stone. Some distance above this stone a second was found impacted with similar perforations. There was a fistula between the gallbladder and the duodenum, but the former had completely discharged its contents, three stones in all." Holz[8]

mentions five cases of recurrent gallstone ileus, in three of these the second stone was removed during the immediate convalescence from the first operation. Van Ravenswaay[11] reported a case in which the obstruction occurred ten days after removing the first stone from the small intestine; rectal examination revealed another stone high up in the rectum. After manual removal, her recovery was uneventful. Schwarke[12] reported recurrence in a sixtynine-year old woman who recovered after her second operation. Wakeley[5] described an obstructive case relieved for eighteen hours by removal of the stone manually from the rectum. After this time a second stone was removed from the ileum and the patient died later of paralytic ileus. At autopsy a cholecyst-duodenal fistula was demonstrated. Hinchey[7] reported the case of a patient in 1940 who expelled a large stone with two facets following a barium enema; a second stone was passed a week later. She was then admitted with an obstruction and an x-ray diagnosis of high intestinal obstruction due to impacted gallstone was made before operation. At operation a stone was removed from the jejunum without searching for an additional stone. She was re-admitted three months later with similar symptoms, the same x-ray diagnosis was made preoperatively and a second stone was removed from the jejunum. The convalescence was stormy but she recovered.

MORTALITY

Balch[6] reported seventeen cases of gallstone ileus; of these ten patients died. Wakeley[5] reported only three deaths in eleven cases. Souttar[15] reported twentyeight cases of gallstone ileus with fourteen deaths, Vick,[16] forty-seven cases with a mortality of 70 per cent, and Courvoisar,[17] 125 cases with a mortality of 44 per cent. In our series, one of three patients died.

CASE REPORTS

CASE I. Mrs. Z. H., sixty-three years of age was admitted November 17, 1941. Six days

previous to this admission there was a sudden onset of upper right quadrant pain, radiating to the right scapula, associated with nausea,

dence of a hernia. Pelvic rectal examination was negative.

Supportive treatment of i.v. glucose was

Fig. 4. Case 11. Routine three-hour plate in gastrointestinal series. Findings were (1) barium in biliary tree and atrophic gallbladder; (2) dilated small bowel. A diagnosis of biliary fistula, high intestinal obstruction and probably gallstone ileus was made.

Fig. 5. Case 11. Routine six-hour plate in same gastrointestinal series as in Figure 4. Findings same as in Figure 4 with visualization of gallstone within the bowel at site of obstruction. The diagnosis was certain for gallstone ileus.

vomiting and intermittent abdominal cramps. There were no jaundice, chills or noticeable fever. The patient had complained for years of intolerance to fatty foods and excessive eructation and flatulence. She had had previous attacks of right upper quadrant pain as far back as twenty years before, and had one severe attack similar to the present one year ago. Her bowels were usually quite regular with no passage of blood, pus and no change of bowel habits up to her admission.

Physical examination showed her temperature to be 99.6°c., pulse 68 respiration 22, blood pressure 137/88, hemoglobin 90 per cent, white blood count 11,000, icteric index 10, and blood sugar 135. The patient appeared more acutely ill than her chart would indicate. Her mucous membranes were dry, her abdomen was soft, lungs were clear, and the heart not enlarged; it was regular in rhythm. There was definite epigastric and upper right quadrant pain on pressure with spasm of the right rectus muscle. There were no palpable abdominal masses, no previous operative scar and no evi-

started and on the second night the patient retained most of the gallbladder dye in spite of the persistent nausea and some vomiting. X-ray examination revealed a nonfilling gallbladder with dilated duodenum and jejunum, gas in the biliary tree and one circular opacity within the lumen of the jejunum that was interpreted to be a gallstone. (Fig. 1.) After ingestion of barium by mouth the stomach and duodenum appeared normal but barium filled the biliary tree. The stone was not seen in these films but the diagnosis remained gallstone ileus.

Since the patient was getting progressively weaker, immediate operation was undertaken to remove the stone. This was done on November 19, 1941. Under sodium pentothal anesthesia (1.25 Gm.) the abdomen was easily entered through the upper right rectus approach. Dilated loops of small bowel were immediately encountered and on withdrawing approximately two feet of this dilated intestine, the point of obstruction was located beyond which there was a striking contrast of collapsed intestine. A hard object could be palpated, presumably a gallstone of more than one inch in

diameter. Without further exploration, the stone was removed via a longitudinal incision, after pushing it down into healthy bowel, a

FIG. 6. Case II. Routine twenty-four-hour plate shows most of the barium in the small bowel but there is a small amount beyond in the colon. The stone can still be seen at the site of obstruction and a Miller-Abbott tube has been inserted into the jejunum.

distance of about three inches. The opening was closed longitudinally with No. 00 chromic catgut, reinforced with fine black silk. Although the lumen appeared constricted to approximately two-thirds the normal diameter, it admitted the forefinger. The gallbladder was now felt for but was so involved with dense adhesions that it could not be definitely identified. The small intestine above the operative site was carefully palpated for an additional stone but none was found. The abdomen was then closed in layers, 5 Gm. of powdered sulfathiazole being sprinkled in the wound. Toward the end of the operation the patient's pulse became rapid and irregular; it was thought that she had auricular fibrillation.

The patient's convalescence was uneventful to her sixth postoperative day. An electrocardiogram had confirmed auricular fibrillation and suggested myocardial damage but her pulse had become for the most part regular. On the sixth postoperative day she vomited a large amount of foul-smelling, yellow material. There was no abdominal distention and no abdominal cramps; the wound was perfectly clean and she was afebrile.

On the eighth postoperative day, because of the persistency of the vomiting and the onset of abdominal cramps in spite of drainage by a Miller-Abbott tube, a flat plate of the abdomen was taken. It definitely showed dilated small bowel and a shadow suggestive of a second gallstone within the lumen. (Fig. 2.) There were apparently more loops involved than in the previous x-rays. Diagnosis was made of recurrent gallstone ileus due to stone in the jejunum below the site of the previous jejunotomy. Nonprotein nitrogen and chloride were 29 and 431, respectively. An emergency operation was performed that night.

Under sodium pentothal anesthesia (0.7 Gm.) the old incision was quickly opened. On withdrawal of about two feet of distended small bowel, the site of obstruction was identified within which there was a hard object about one inch in diameter and beyond which there was collapsed bowel. The remaining twenty odd feet of distal small bowel was now delivered and palpated for the presence of an additional stone, but none was found. After this, the dilated portion of the bowel was palpated up to the site of the previous operation. At this point there was a rather firm adherence between the omentum and the suture line which was apparently causing no obstruction. It was thought wisest not to release these adhesions, so the remaining two to three feet to Treitz's ligament were not examined. The gallbladder area was again palpated without positive findings due to the adhesions. The stone was apparently in the midjejunum; it was milked down about three inches into normal bowel with some difficulty. It was removed via a transverse incision which was closed transversely with a continuous suture of No. 00 chromic catgut, reinforced with interrupted mattress sutures of fine black silk. The lumen was very slightly constricted and easily admitted the forefinger. It should be mentioned that the distance between the two jejunotomies was approximately three feet, and that a distance of at least two feet below the first operative site was examined at the first operation, but no farther because of the collapsed character of the bowel. Again 5 Gm. of powdered sulfathiazole was sprinkled into the

abdominal wound, a small portion being smeared over the suture line, the wound was closed with silver wire sutures. The total time of operation was about fifty minutes. The convalescence was essentially uneventful, the wound healing per primum.

A check gastrointestinal series, on December 19, 1941, gives a clear demonstration of the persistence of the biliary fistula at least a month after discharge of the two stones. (Fig. 3.)

CASE II. Mrs. A. E., sixty-five years of age, was admitted on April 15, 1941. Her symptoms began two weeks previous to admission with pain in the upper midabdomen radiating to the middle of chest. There were no jaundice or constipation. Nausea and vomiting occurred three days previous to admission initiated by sudden onset of severe cramping pain in the right subcostal region and radiated to the right scapula. There were no chills or noticeable fever. She had had recurrent attacks of gallbladder disease.

On physical examination the abdomen was not distended. There was moderate tenderness of the subcostal region with slight spasm and rather marked tenderness over the entire left lower quadrant. No masses or hernia were found. Pelvic rectal examination was essentially negative. Her temperature was 99°C., pulse 86, respiration 22, blood pressure 122/68, hemoglobin 88 per cent, nonprotein nitrogen 71, sugar 140.

Vomiting persisted for the first nine days in the hospital so that intravenous therapy was practically her only source of nourishment. She appeared weaker but a gastrointestinal series taken on April 24, 1941 revealed a high intestinal obstruction with barium in the biliary tree and an oval shadow, apparently a stone, within the lumen of the intestine. (Figs. 4, 5 and 6.)

A diagnosis of gallstone ileus was made due to the presence of a gallstone in jejunum. In spite of intravenous therapy and gastric lavage the patient's course was downhill. Nonprotein nitrogen was 87 and there was a 3+ acetone in the urine.

On April 25, 1941, under ether anesthesia, a right rectus incision was made and the abdomen easily entered. The distended small intestine was traced distally to a point at the terminal portion of the jejunum where a large gallstone was found producing obstruction.

This was removed with a transverse incision closed with a continuous mattress suture of No. 00 chromic catgut, reinforced with two layers of interrupted Lambert sutures of fine black silk. The lumen admitted the tips of two fingers. The liver felt perfectly normal. There were dense adhesions about what appeared to be the gallbladder and the adjacent duodenum. The omentum also enveloped these structures. No other stones were palpable in this area, but there was no systematic search of the entire length of small gut either above or below the obstruction. No attempt was made to free the adhesions to demonstrate the connection between the duodenum and the gallbladder. The abdomen was quickly closed with the patient in good condition.

Except for a low grade infection of the wound the patient made an uneventful recovery and was discharged on the nineteenth postoperative day.

A check x-ray one month after operation revealed barium in the biliary tract indicating persistence of a biliary fistula. Three months after operation, the patient was admitted with abdominal pain most marked in the right upper quadrant associated with nausea and vomiting and slight jaundice. A gallbladder series taken at this time revealed a nonfilling gallbladder. After a few days of conservative treatment, the symptoms subsided and the patient was discharged.

Eight months after operation, the administration of barium by mouth revealed no evidence of a fistula. She has since enjoyed good health.

CASE III. The third case, Mrs. A. N., was a fifty-three-year old white female, admitted October 25, 1939, with a diagnosis of acute mechanical obstruction and diabetes mellitus. (One member of the house staff suggested gallstone ileus.) On x-ray a flat plate of the abdomen confirmed the diagnosis of obstruction only. At operation the gallbladder was buried in dense adhesions and except for definite distention the upper intestinal tract was normal. There was marked edema and inflammatory reaction around the ileocecal valve. A stony object was identified in the transverse colon and milked down into the rectum. (This object was removed two days later on rectal examination and identified as a gallstone.) No other pathological condition of the remaining viscera could be demonstrated. After a stormy post-

operative course of two weeks, complicated by exacerbation of the diabetes, the patient died of generalized peritonitis.

DISCUSSION

The extremely high mortality demands early treatment. This becomes feasible only after diagnosis has been established. When nausea and vomiting are out of all proportion to other symptoms of gallbladder disease, gallbladder ileus should be considered and x-ray examination made. Evidence of intestinal obstruction from dilated intestine alone would justify immediate exploration; but if the biliary system is filled with gas or a stone can be seen within the bowel, exploration becomes imperative.

After locating the stone, a thorough search of the bowel above and below should be done if the patient's condition permits. Collapse of the bowel distal to the stone does not exclude the possibility of a second one below. This was experienced in the first case reported in which the second smaller stone had apparently produced only partial obstruction and was masked by the three feet of collapsed bowel proximal to it. That this stone could have passed the narrowed lumen at the suture line after the first operation is most unlikely. We believe that it could have been manipulated in a retrograde direction and removed from the original intestinal opening had it been found. When a large facet is noted on a gallstone, one should account for the second stone to avoid possible recurrence of ileus.

An occasional complication arises from diminution in the size of the lumen of the bowel due to method of closure. A longitudinal incision is recommended with a transverse closure, thus leaving the largest possible lumen.

REFERENCES

1. MARTIN, FRANK. Intestinal obstruction due to gall stones. *Ann. Surg., 55:* 725, 1912.
2. BORMAN and RIGLER. Spontaneous internal biliary fistula and gall stone obstructions. *Surgery,* 1: 349, 1937.
3. JUDD, E. S. Internal biliary fistula. *Surgery,* 81: 305.
4. WAKEFIELD, VICKERS and WALTERS. Intestinal obstruction caused by gall stones. *Surgery,* 5: 670.
5. WAKELEY, WILWAY. Intestinal obstruction by gallstones. *Brit. J. Surg.,* 23: 377, 1935.
6. BALCH, F. Gall stone ileus. *New England J. Med.,* 218: 11, 1938.
7. HINCHEY. Recurrent gall stone ileus. *New England J. Med.,* 218: 11, 1940.
8. HOLZ, EDMUND. Zur Frage der Gallensteinileus. *Arch. f. klin. Chir.,* 155: 166–176, 1929.
9. PYBUS, A note on two cases of gall stone ileus. *Lancet,* 2: 812, 1922.
10. MOSCHOWITZ. *Ann. Surg.,* 61: 242, 1915.
11. VAN RAVENSWAAY. Acute intestinal obstruction by large gallstones. *Am. J. Surg.,* 16: 56, 1931.
12. SCHWARKE. U Zweimaligen Gallensteinileus. *Zentralbl. f. Chir.,* 65: 1980–1985, 1925.
13. DREW. *Brit. J. Surg.,* 12: 803, 1925.
14. RIGLER. Gallstone obstruction. *J. A. M. A.,* 117: Nov. 22, 1941.
15. SOUTTAR. Discussion on acute intestinal obstruction. *Brit. M. J.,* 2: 1000–1001, 1925.
16. VICK. Statistics on acute intestinal obstruction. *Brit. M. J.,* 2: 546–548, 1932.
17. COURVOISIER. Beitr. Z. Path. u. Chir. der Gallenweger. Leipsig, 1900. Vogel.
18. POWERS. Acute intestinal obstruction due to impacted gall stones. *Surg., Gynec. & Obst.,* 47: 416, 1926.

A NEW TYPE OF RELAXING INCISION—
THE DERMATOME-FLAP METHOD*

Henry N. Harkins, m.d.

Diplomate of the American Board of Surgery; Associate Surgeon, Henry Ford Hospital

DETROIT, MICHIGAN

THERE are three methods of making a relaxing incision in common use at the present time: (1) Leave the incision open to heal in by third intention; (2) leave the incision open to be skin grafted at a later date, and (3) primary skin graft from a remote site.

Each of these methods is open to some criticism. The first is a deforming procedure producing a raw open wound which may take months to heal. The second prevents much of the deformity, but requires two separate operative procedures. The third method is advised by Davis (1919). While this latter technic has many advantages over the other two it produces three separate wounds (primary operative incision, relaxing incision, and skin graft donor site). The use of relaxing incisions is an important adjunct to many surgical operations, for example, the removal of large superficial tumors or excision of osteomyelitic scars of the tibia. It is quite possible that if relaxing incisions were simplified, their increased use with resulting decrease in tension of the primary wounds would greatly facilitate healing of the latter in many difficult cases.

The method devised by the author and used in the following illustrative case has the advantage that the relaxing incision is made in the skin graft donor site, eliminating the necessity for a separate wound to obtain the graft.

CASE REPORT

J. W., male, aged twenty, had a large hemangioma of the skin and subcutaneous tissue of the anterolateral surface of the left thigh measuring about 15 by 35 cm., shown in Figure 1. This tumor had been present for many years, and interfered to some degree with the patient's walking, but, more urgently, with his induction into the armed forces.

Fig. 1. Hemangioma of anterior surface of left thigh.

Operation. A 30 cm. long elliptical excision was made on March 17, 1942, removing as much as possible of the large hemangioma which was found not to extend below the muscular fascia but to be connected with the latter at several points. It was found that the defect remaining was so large that it could not be closed without great tension, so a new type of relaxing incision was made on the anteromedial surface of the left thigh. A 20 cm. long dermatome graft averaging a little less than 6 cm. in width was cut from above downward using the Padgett-Hood dermatome. The dermatome was set to cut a graft of 0.012 inches in depth. The graft was left attached at its lower end and reflected inferiorly as shown in Figure 2. A vertical relaxing incision a little over 2 cm. deep was then made in the center

of the donor area, also as shown in Figure 2. This spread to an average width of 2.5 to 3.0 cm., relaxing the tension on the primary wound sufficiently to allow closure of the latter. The

Figure 4 shows the condition on that day just before stitch removal. The condition ten weeks after operation is shown in Figure 5, demonstrating good healing.
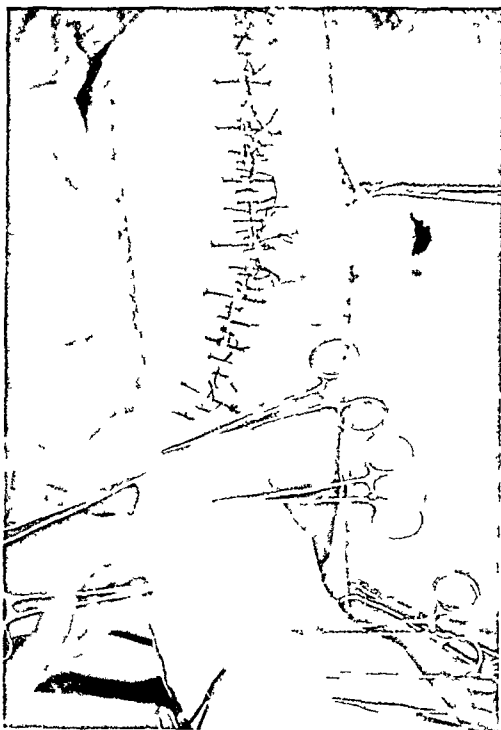
FIG. 2. Second stage of dermatome-flap relaxing incision The hemangioma has been excised leaving the stitched incision on the right hand side of the figure. The dermatome-flap graft is turned down on the gauze. The relaxing incision has been made in the bed of the raw donor area. The graft is still attached at the lower end of the donor area thus producing the flap
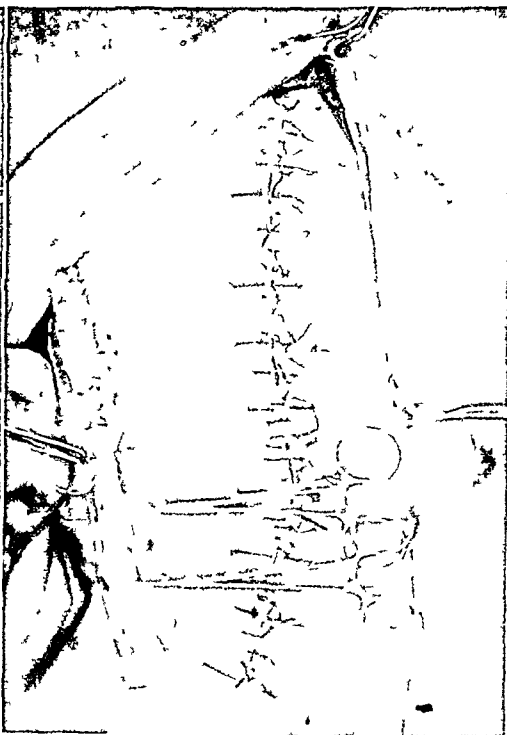
FIG 3 Third stage of dermatome-flap relaxing incision. The graft has been turned back into the central portion of its bed and is being sutured with interrupted silk stitches into the relaxing incision.

graft was then sutured down into the defect made by the relaxing incision with numerous interrupted sutures, a portion of which is shown in Figure 3. Ten Gm. of sulfanilamide powder were then sprinkled over the graft and surrounding bare donor area. This was covered with fine mesh vaseline gauze, mechanic's waste and dry gauze and then the entire thigh wrapped with elastoplast, the primary incision on the lateral surface of the thigh having been covered with dry gauze.

Pathologic Examination. Sections revealed a cavernous hemangioma.

Postoperative Course. The first dressing of the primary incision was done on the ninth day after operation and the stitches were removed from the graft on the fourteenth day.

COMMENT

With the introduction of the Padgett-Hood dermatome skin grafting has become so much easier that new opportunities for perfection of surgical procedures are offered. The present operation is an example in kind and simplifies the use of relaxing incisions. The only difficulty that is apt to be encountered with this technic is that the very tautness of the skin which interfers with the closure of the primary wound is apt to hamper the taking of the dermatome graft. Razor grafts are most easily taken when the skin is taut, dermatome grafts when it is relaxed. For this reason, any sutures in the primary wound should not be tied until the graft is taken. An example of this is shown by the follow-

ing account: On May 21, 1942, i.e., two months after the original operation, the remainder of the hemangioma was removed

has the following suggestions for carrying out the technic of this procedure:

1. *Selection of a Donor Area.* With the
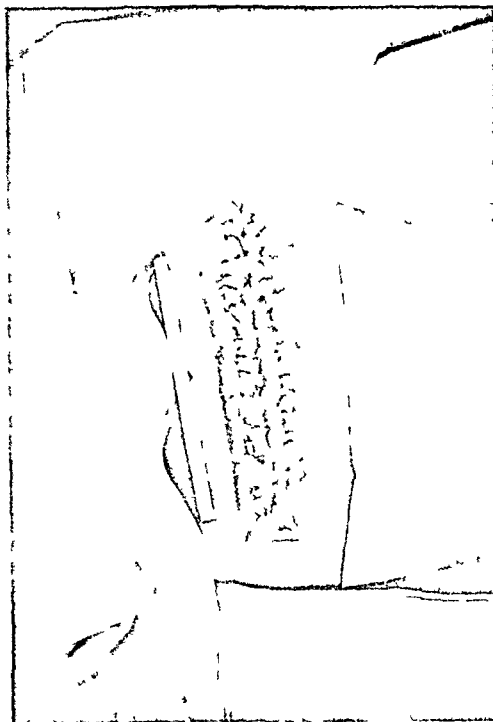
FIG. 4. The relaxing incision fourteen days later. The stitches were removed immediately after this picture was taken and the graft was found to be over a 98 per cent take. The attachment of the flap at the lower end of the defect is seen while the already healed but ungrafted lateral portions of the donor site are readily visible.

FIG 5. Result ten weeks later. The depression left by the relaxing incision and covered by the graft is now practically flush with the surrounding skin. The wound and relaxing incision are entirely healed

from the thigh of patient J. W. A dermatome-flap relaxing incision was attempted on the posterolateral surface of the thigh. The skin was so taut, however, despite the fact that the primary wound was not closed, that no satisfactory graft could be cut. Therefore, an ordinary relaxing incision was made with primary closure by a dermatome graft from the opposite thigh. This resulted in an over 95 per cent immediate take.

TAKING OF DERMATOME GRAFTS

With a personal experience of taking over sixty dermatome grafts, the author

Padgett-Hood dermatome, practically any portion of the body is available for furnishing a graft, whether that area be concave or convex. The abdomen and thighs are most suitable.

2. *Preparation of Donor Area.* This is best done with alcohol followed by ether. The use of certain other antiseptics interferes with the adhesion of the glue.

3. *Adjustment of Dermatome.* The depth of the graft should be 0.012 to 0.020 inch for the average adult, while in children or those with atrophic skins it should be less than the average figure. For a child two to five years of age a depth of 0.007 to 0.009 inch is satisfactory. Thinner grafts are best for covering granulating surfaces, while thicker ones are best for clean cases.

4. *Application of Glue.* The glue should be put on as a thin sheet without bare spots. A tongue depressor or the base of the dermatome is useful for applying the glue uniformly. It should be painted on both skin and drum surface and allowed to dry for at least sixty seconds.

5. *Taking of Graft.* The donor area should be adjusted so that the skin is relaxed as much as possible. This is exactly the opposite from what is necessary when razor (Thiersch) grafts are taken, in which case the skin is held tautly by boards or an assistant. This difference is important and well meaning assistants should be waved aside. Taking of a dermatome graft is best a one man job. If the desired graft is to be narrower than the width of the drum, it is far easier to apply the glue uniformly and then rub off the areas not to be included than to attempt to spread the difficultly manageable wet glue according to any pattern. The graft is taken in the usual manner with a sawing motion. For application of a dermatome flap graft, the end of the graft is left attached. It is not for a moment considered that this attachment furnishes nutrition to the graft; the latter is merely left intact to facilitate subsequent resuture.

6. *Suture of Graft.* This is best done meticulously with numerous interrupted fine silk sutures. These should be applied both around the edges and across the bed of the graft, this latter step being particularly important for a dermatome-flap relaxing incision in which instance the graft is, of necessity, applied to a concave surface. Since the relaxing incision takes up most of the width of the graft, the rest of the donor site is left bare to heal in the usual manner, which is usually before the time of the first dressing.

7. *Dressing of Graft.* The combined grafted area and uncovered donor site are covered with a fine sprinkling of sulfanilamide powder, then by fine mesh vaseline gauze, mechanic's waste, and finally a dry dressing covered by elastic adhesive. The groove left by the relaxing incision is packed with a moulded piece of mechanic's waste to produce uniform pressure.

8. *Subsequent Care.* Stitches are removed on the fourteenth day at which time the first dressing is changed. The healing of the bare donor area is best accomplished by not changing the dressing until the fourteenth day. Since this prevents removal of the stitches in the grafted area until that time, this might be considered a theoretical disadvantage of the procedure. No difficulty was encountered in the case under discussion, however, and stitches left in other skin grafts for this period of time also led to no difficulties.

SUMMARY AND CONCLUSIONS

A new method of making a relaxing incision is presented. This combines the advantages of primary closure by a skin graft with the lack of necessity for making a separate wound to obtain closure.

The dermatome-flap relaxing incision consists briefly in making a dermatome graft which is left attached at one end and reflected back. The relaxing incision is then made in the donor area or bed of the graft and the flap turned down again to cover the raw area made by the incision.

This method is applicable to all situations in which relaxing incisions are useful. The latter are of especial advantage for wounds of the extremities in which large superficial tumors or scars are excised.

REFERENCE

1. DAVIS, J. S. Plastic Surgery: Its Principles and Practice. Philadelphia, 1919. P. Blakiston's Son & Co.

CONTROL OF SOMATIC PAIN*

WILLIAM BATES, M.D.

PHILADELPHIA, PENNSYLVANIA

THE relief of pain has always been one of the chief functions of a doctor. Pain of visceral origin has been the reason for the existence of a surgeon. However, the differentiation between visceral and somatic pain has not always been an easy one to make.

The types of somatic pain to which we have devoted most of our attention are those neuralgias which are segmental in distribution and which are associated with tenderness. Pain due to segmental neuralgias, by virtue of its variation in location, character and intensity, may simulate the pain of almost any form of visceral disease.

Evaluation of the pain in these patients has been made more definite by various factors: (1) The finding of tender points at the emergence of the posterior, lateral or anterior cutaneous branches of the intercostal nerves; (2) the establishment of tenderness in segments corresponding to definite dermatomes which are associated with the pain; (3) the fact that segmental tenderness is rarely initiated by the pain of visceral stimuli, unless a widespread peritonitis is present; (4) the finding of reduced skin surface temperature which is often associated with the pain and tenderness, and (5) following nerve block, pain and associated tenderness should disappear simultaneously.

In many series of operations, the failures reported are chiefly in patients whose cardinal complaint was pain. This I believe is not due to incomplete or incompetent surgery but to failure in diagnosing the source of the pain.

In 1926, when a former member of the Academy of Surgery, the late Dr. J. Berton Carnett, first wrote about intercostal neuralgia of the abdominal wall, he stressed this same fact.

His work met with differences of opinion, but his basic reasoning has been substantiated many times. It is very true that to follow his teachings of that time might have led to some serious mistakes. On the other hand, it would have prevented many useless operations. In his teachings, he advocated differentiating visceral from somatic pain. He described his tests to accomplish this. He gave a series of causes for the existence of somatic or as he called it, intercostal pain, and lastly, he attempted to outline methods of treatment for this condition.

This latter fact was unfortunate and was forced upon him and his staff rather prematurely. Having brought the picture of surface pain to the attention of practitioners they demanded some method of treatment. Among the recommendations for treatment was, of course, the classic one of "eliminate the cause." This we tried to do, but in many cases we found that the pain persisted long after the apparent cause had been removed. Something more was needed in those patients whose toxic foci were eliminated and postural defects corrected, with no relief of their pain.

At this stage of development of our clinical problem, an attempt was made in cases of local pain and tenderness to inject with novocain. As one might expect, most of the cases got only temporary relief. However, quite a few got prolonged relief encouraging us to repeat the injections. When prolonged relief was obtained by such a simple procedure, we were of the opinion that the basic cause or causes were probably corrected in conjunction with the infiltration.

About 1931, one of the men on Dr. Carnett's service at the Graduate Hospital, Dr. B. D. Judovich, did some work on the

* Annual Oration for 1941. Read before the Academy of Surgery, November, 1941, Philadelphia.

83

pitcher plant—Sarracenia purpurea. He prepared an aqueous solution derived from this plant, and observed that it was of value in relieving pain of neuralgic origin. It was believed at that time that its properties were due to the presence of amines. It was noted that the preparation had an action upon sensory nerves, relieving neuralgic pain without producing changes in skin sensation and having no effect upon motor nerves.

Dr. Carnett suggested that Dr. Judovich be allowed to work in what we called the Intercostal Neuralgia Clinic. Heretofore we had been trying to determine the cause of the pain, and treat by means of eliminating focal infections, correction of postural defects, giving Goldthwaite exercises and the occasional injection of novocain solution in some of the more persistent cases of localized pain and tenderness.

From then on, even though we did not know the actual active principle of the solution we called Sarapin, it was administered to numerous patients suffering with pain of somatic origin.

Controls of novocain, saline and water were used, and the results recorded. The key numbers of these various ampoules were changed several times, and on analysis in each series, it was found that Sarapin produced prolonged relief in contrast to fleeting or negative results with the other solutions. In a number of instances, patients who had been injected with novocain with only a short period of relief of pain, obtained prolonged relief by a subsequent injection pitcher-plant distillate.

Toxicity tests revealed that it was harmless. It caused no tissue coagulation nor sclerosis. This action was unusual and difficult to substantiate. We were treating a subjective symptom, and laboratory animals gave no actual data except in reference to toxicity. Routine pharmacologic tests in three different laboratories threw no light upon the problem.

Following its injection of peripheral nerves, there were no cases of motor weakness, nor loss of touch, pressure, pinprick nor temperature sensibility. In some cases, one infiltration of the distillate was sufficient to provide permanent relief of pain, even though of long duration. The psychic factor of relief in this type of pain has largely been ruled out by establishing the presence of tenderness in the dermatomes of the painful segments.

In intercostal neuralgia, paravertebral injection of the nerve trunks involved, results in an immediate intensification of the pain, followed during the next thirty minutes, by a gradual contraction of the hyperalgesic area in the dermatome supplied by the nerve. In a large number of cases it is followed by complete relief of neuralgia.

Because of the segmental overlap, the effect of the distillate upon sensations other than neuralgia cannot be evaluated.

If, in a case of sciatic neuralgia, the region of the sacrosciatic foramen is infiltrated with the pitcher-plant distillate, there is relief of the neuralgic pain as well as of tenderness along the nerve trunk. This is similar to results from infiltration of novocain, but in contrast to the action of novocain there is no numbness, no loss of sensibility and no motor weakness. Reflexes remain unchanged.

In 1939, investigation was made by Dr. Winifred Stewart and Dr. Joseph Hughes, using the cathode ray oscillograph to determine the action of the pitcher-plant distillate upon the nerve impulse.

Their studies indicated that the somatic c wave impulse, which is supposedly transmitted by way of the small unmyelinated fibers, was depressed in each instance. A report of these findings was made in 1940 by Stewart, Hughes and Judovich in which they stated: "The ability of pitcher plant distillate to abolish neuralgia pain which; like fascial pain, is aching, poorly localized, and frequently associated with nausea and sweating, and at the same time, to leave pinprick as well as other forms of sensibility unaffected, led us to inquire further into its physiological action, and to attempt to determine its active principle.

"The effect of pitcher plant distillate on the action potentials of the saphenous nerve of the cat was observed. The nerve was mounted in a nerve chamber in a gas mixture of five per cent carbon dioxide and ninety-five per cent oxygen. The temperature was maintained at 37.5° to 38°c. The nerve was so mounted that it could be bathed in the solution to be studied. The pitcher plant distillate was adjusted to a pH. of 7.4. The action potentials were recorded on a cathode ray oscillograph.

"After five minutes immersion in pitcher plant distillate, the maximal A spike was somewhat reduced while the c fiber potentials were obliterated."

For the first time it appeared that we had some definite objective proof that the solution we had been using should relieve pain. It became vitally important to us to know the chemical constituents of this solution. The first report we received on the analysis showed that the crystalline compound which was isolated was ammonium chloride. Experiments in the laboratory were carried out with distillate of pitcher-plant solution of known ammonium chloride. All three solutions gave the same result. Clinically, injections of Sarapin and of a solution of ammonium produced the same prolonged relief.

On checking the chemical process by which the distillate was obtained, Judovich found that the end result should have been a sulfate and not a chloride. Accordingly, further experiments were carried out with a solution of known ammonium sulfate. Both clinically and experimentally the results were parallel to those found with the solution of ammonium chloride. From this it was readily deducted, that, as the ammonium radical was only constant, that it was the active principle responsible for our results.

These observations on actual patients led us to believe that neuralgic pain and pinprick are mediated by separate fiber groups. Clinically, however, we have been unable to relieve pain of visceral or sympathetic origin by the injection of ammonium salts.

In two instances the first and second lumbar sympathetic ganglia were injected in a patient with vascular occlusion of the lower extremity. There was no change in the skin surface temperature of the leg following infiltration of these points with ammonium salts. The needles were left in place and at the end of twenty minutes 2 per cent novocain solution was injected. Within two minutes a definite rise in skin temperature was obtained. All patients benefitted by these injections were cases of pain of somatic origin associated with tenderness.

On occasion, the infiltration of the pitcher-plant distillate and the ammonium salts have been of value in differential diagnosis; neither of these substances has any effect upon vascular pain or pain of visceral origin.

Just what the difference is in the unmyelinated c fiber of peripheral distribution which responds to these injections, and the unmyelinated c fibers of the sympathetic nerves and their ganglia which do not respond to these injections, we do not know. Experiments are being conducted by Dr. Stewart and Dr. Hughes upon sympathetic fibers to determine the effect of ammonium salts upon the nerve impulse in this type of tissue.

This has no doubt been a prolonged explanation of a simple procedure, but the clinical results have been so satisfactory to some of us, that the confirmation of our ten years of clinical observations by laboratory proof, make it seem worth while to render this report.

Our work in attempting to control pain is by no means complete, as the same solutions are now being tested intraspinally for intractable pain. To date the results in certain cases have been most promising, but more data must be collected before definite conclusions can be made.

I am not recommending regional infiltration as a cure-all for aches and pains, but without it, we would have been unable to

relieve many patients of their persistent pain.

SUMMARY

1. Segmental neuralgias may often simulate the symptoms of visceral disease. These very often account for pain which persists following surgery.

2. Various factors are mentioned which are important in the differentiation of pain of visceral and pain of somatic origin.

3. Regional infiltration appears to be the most efficacious method of treatment for somatic pain. It is also of value in diagnosis.

4. The use of the pitcher-plant extract, in many patients, has afforded prolonged relief as compared with procaine.

5. Injection of solutions of ammonium inorganic salts both locally and intra-spinally have given promising results. The advantages are nontoxicity and absence of tissue damage.

6. A résumé is given of the experimental and clinical value of both the pitcher-plant and ammonium salts.

OCCASIONALLY a patient will receive an overdose of a local anesthetic agent and have convulsions or convulsions may develop under general anesthesia. If the convulsions are severe, death will ensue unless artificial respiration is carried out to ventilate the patient enough to support life. The use of an anticonvulsant drug may be necessary to accomplish this. From "Clinical Anesthesia" by John S. Lundy (W. B. Saunders Company).

THE CURATIVE TREATMENT OF HEMORRHOIDS*

LIEUT. COMMANDER EMIL GRANET, M.C., U.S.N.R.

GUANTANAMO BAY, CUBA

INASMUCH as the anorectal region is easily accessible to examination and treatment, the patient afflicted with hemorrhoids expects from his physician and is entitled to permanent cure. These are to be accomplished with a minimum loss of working time and little or no pain. These desiderata can and should be attained. Too often they are not.

The term, hemorrhoids, refers only to varicosities of the veins of the hemorrhoidal plexus. Internal hemorrhoids occur proximal to the anorectal junction and external hemorrhoids distal to this line. Hemorrhoids may remain innocuous for years. They cause symptoms when they become the seat of further pathological changes incident to trauma and infection. Bleeding from internal hemorrhoids usually results from laceration of the varix by the trauma of defecation. Prolapse and partial strangulation of an internal hemorrhoid results in perianal soiling with bloody mucus, pruritus and occasionally pain. Pain associated with hemorrhoids is due to inflammatory edema resulting from mucosal fissuration, thrombus formation or strangulation. Wholly or in part these symptoms finally force the patient to seek relief, too often after a long course of fruitless self-medication with salves, suppositories and numerous patent pile remedies.

SCLEROTHERAPY

Freedom from pain and loss of working time, as well as cost of hospitalization makes the sclerotic injection treatment of hemorrhoids desirable. The effectiveness of sclerotic injections depends upon the fibrosis resulting from the inflammatory reaction induced by the deposition of a chemical into the submucosa above, or into the lumen of the hemorrhoid The relative technical simplicity of the method makes it available to the general practitioner who is not necessarily a skilled surgeon. Unfortunately, permanent cure can be attained in only a small percentage of patients. Sclerotherapy is permanently effective only in those instances in which hemorrhoids are internal, small in size and number and not associated with mucosal prolapse. Such ideal cases are rarely seen clinically because at this stage symptoms are few and infrequent. Sclerotherapy is valuable in the treatment of large hemorrhoidal varicosities to stop hemorrhage temporarily until operation can be performed. It also has its place in allaying symptoms caused by hemorrhoids in cardiacs, nephritics, diabetics, the senile, and in all conditions in which operation is hazardous or impractical. When injection therapy is administered, it is essential to follow and examine patients periodically for years, as it will be found that recurrences of new varicosities requiring further injection commonly occur in the interval since previous treatment. Often patients who have been injected for years eventually submit to operation because of the discomfort resulting from increasing mucosal prolapse which cannot be further controlled by injection.

The technic of injection, indications, contraindications and dangers, have been described by Buie,[1] Gabriel,[2] Hirschman,[3] and Swinton.[4] My personal preference in treating properly selected cases is the use of phenol and glycerin, each 5 per cent in water, injected through a Hirschman-type anoscope using a No. 23 gauge angulated tonsil anesthesia needle. In my experience, the injection of 5 per cent quinine and urea caused inordinate pain despite proper technic, and slough occa-

sionally resulted in quinine sensitive patients. Almond, and later peach kernal oil as diluents for phenol were discarded because of the occasional occurrence of eleomas, benign fatty tumors in the submucosa at the site of the injection. These tumors can coalesce to cause partial stricture (Rosser[5]).

Sclerotherapy, in short, can cure hemorrhoids in a limited number of early cases and is a valuable palliative measure to control hemorrhage. When utilized, its temporary efficacy must be clearly explained to the patient, emphasis being placed on the necessity for continued observation and possible re-injection for years.

HEMORRHOIDECTOMY

Physiological limitations of sclerotherapy often result in recurrent hemorrhoidal lesions. It is difficult, however, physiologically to account for the all too frequent "recurrence" of hemorrhoids and mucosal prolapse after purported surgical excision. The term, "recurrence," in this connection is a misnomer. A varix which has been excised cannot recur. Hemorrhoids which occur following hemorrhoidectomy must be due to physiological enlargement of varices and prolapsed mucosa left intact after an inadequate, incomplete operation. These, not infrequent, operative failures may result from carelessness or inexperience on the part of the surgeon.

Its safety from mortality as well as the technical ease of performance relegates hemorrhoidectomy to the category of minor surgical procedures. The busy surgeon usually schedules hemorrhoidectomy as the final case after several tedious, prolonged, arduous laparotomies. He performs this operation when tired, irritable, and perhaps overdue for office consultations. Under such circumstances, I have seen surgeons rapidly overdilate the sphincter muscle; the three primary hemorrhoids are next hurriedly clamped, excised, and the stump sewn over, often including part of the subcutaneous sphincter muscle in the ligature. Secondary hemorrhoids are disregarded, either from neglect or from fear of excessively denuding mucosa. The excessive perianal edema and pain which frequently follow such operations are due to sphincter spasm resulting from the trauma of overdilation and from strangulation of muscle fibers included in the hemostatic ligature. Failure to remove the minor secondary hemorrhoids results in physiological enlargement of these varicosities with early return of symptoms. The surgeon is confronted only too soon with an irate and dissatisfied patient justifiably accusing him of incompetence in performance of the operation.

It is the exceptional patient who submits to operation with only three primary hemorrhoids involved. After many years of self-medication and perhaps an unsuccessful course of sclerotic injections, the patient usually comes to operation presenting a veritable ring of prolapsed ulcerated, internal hemorrhoids, hypertrophied papillas, mucosal fissuration and numerous skin tabs. Faced with such a formidable array of lesions, the inexperienced surgeon fearing postoperative stricture as a result of excessive tissue loss, wisely concludes that discretion is the better part of valor and removes only the larger varicosities. Revision operations in such cases are frequent.

Inadequate hemorrhoidectomies will continue to be performed until the general surgeon shows willingness and interest to apply the newer concepts of anorectal anatomy described by Levy[6] in this country and Milligan,[7] in England, to their technic of hemorrhoidectomy. Many surgeons are slow to utilize measures recently developed which enhance the postoperative comfort of hemorrhoidectomized patients. The laity and physicians alike recall the frightful pain and the discomfort of prolonged obstipation which followed hemorrhoidectomy by the now obsolete clamp and cautery method and the barbarous "whistle" plug necessitated for hemostasis.

One extremely important point should be emphasized: Frequently, a patient complains of rectal bleeding and undergoes hemorrhoidectomy. However, bleeding persists after operation. Eventually a belated digital and sigmoidoscopic examination reveals the primary cause of bleeding to be a neoplasm of the rectum or sigmoid. Competent rectosigmoidal examination by means of the electrically lighted sigmoidoscope infallibly must be conducted previous to any anorectal operation. By this means rectal malignancy can be discovered in the early resectable stage. Physicians can no longer shirk their responsibility to diagnose cancer of the lower bowel early, especially when rectal bleeding is a prominent symptom.

Much has been written on the technic of hemorrhoidectomy and it may be perhaps presumptious to offer still another note on this subject. Competent general surgeons, however, do not attain uniformly satisfactory results in their hemorrhoidectomies as is attested by the too frequent revision operations for "recurrences" and proctotomies for stricture. The postoperative course of their patients is needlessly painful and hospitalization is prolonged. The procedure to be described was designed to avoid these disadvantages, to minimize debility and excess nursing care, and to enable the patient to resume his work in about eight days.

ANESTHESIA

Preoperative preparation is simple. The patient is admitted the evening before operation; an effectual soapsuds enema is administered, following which the patient is shaved. Adequate sedation is administered to insure sound sleep. On arising a pint of warm saline solution is instilled into the rectum and immediately expelled. Rectal lavage is repeated until the return is clear. Pentobarbital sodium, 0.2 Gm., is administered two hours before operation.

Caudal epidural block with 1 per cent procaine solution affords ideal anesthesia for at least one hour. Failure of anesthesia,

3 per cent in my most recent series, is attributable to anatomical deformities which prevent deposition and retention of the medication in the caudal epidural space. For anal surgery, successful caudal block is ideal because its use avoids the nausea, vomiting and special nursing care associated with recovery from inhalation anesthesia, or the headache, nausea and vomiting which followed spinal anesthesia in 30 per cent of the patients operated upon by Jennings and Karabin[8] for anorectal lesions.

With the patient prone on the table in the inverted jack-knife position, 60 cc. of 1 per cent procaine solution is deposited in the caudal canal by the usual technic (Labat[9]). The time interval required for effective anesthesia is about fifteen minutes. Any actual loss of time can be avoided by completing the administration of the anesthesia before the surgeon scrubs. If the surgeon himself performs the nerve block, gloves are worn for this procedure which is then followed by proper scrubbing and draping. If block anesthesia is unsuccessful, local infiltration anesthesia is administered.

OPERATION

The comfort of the patient, surgeon and assistant during operation is enhanced by maintaining the patient in the prone, inverted, jack-knife position. The buttocks are separated by wide adhesive straps attached thereto and thence to the frame of the table. After painting and draping, the anal sphincter is found to be relaxed and easily admits three fingers. Use of the ingenious ring retractor devised by Hullsiek,[10] affords incomparable exposure and dispenses with a second assistant. (Fig. 1.)

The so-called "ligature operation" fulfils all the requirements of a satisfactory operation for hemorrhoids. This operation, in principle, was originated one hundred years ago by Mr. Frederick Salmon, founder of the St. Mark's Hospital for Rectal Diseases in London. It has been used continuously to this day at that institution by such masters of surgery as Allingham, Lockhart-

Mummery, Gabriel and Milligan. In a recent paper Milligan[7] described in detail modern concepts of the surgical anatomy of the anal canal emphasizing its relation to
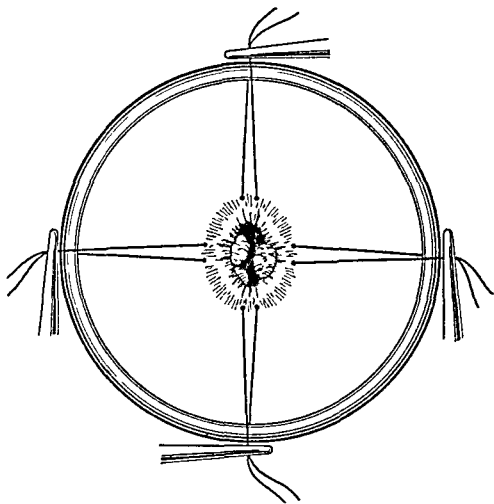
FIG. 1. Exposure of the operative field by means of the Hullsiek anal retractor.

the operative treatment of hemorrhoids. These anatomical principles have been followed in the operation herein described.

After exposure by traction sutures, the anorectal region is carefully inspected and the operative sequence planned. The largest internal hemorrhoid is grasped in an Allis clamp and prolapsed to expose its proximal attachment, the mucosal pedicle. The mucosa is incised parallel to and very close to the base of the hemorrhoidal pedicle bilaterally. (Fig. 2.) The analogus external hemorrhoid or skin tab is now secured by a second Allis clamp and the hemorrhoidal mass is elevated by gentle traction. A small superficial incision is made through the perianal skin about 1 cm. below and parallel to the anocutaneous margin. (Fig. 3.) The thin fibers of the corrugator cutaneous muscle are now seen traversing the wound. These fibers are carefully severed and with blunt, knife-handle dissection directed proximally, a line of cleavage is exposed which leads into the perianal space. The subcutaneous external sphincter, a thin band of smooth muscle, is thereby exposed. This muscle is retracted laterally and further blunt dissec-

tion directed proximally next uncovers the distal portion of the fibrous longitudinal muscle of the rectum. Keeping close to the base of the hemorrhoidal pedicle, incision three is extended proximally to join incisions one and two. Care in dissection is rewarded by absence of free bleeding. The hemorrhoidal pedicle now swings free and contains only hemorrhoidal varicosities, redundant rectal mucosa and skin tabs.

A 30 cm. strong, heavy silk or cotton suture threaded double on a curved hernia needle is passed through the fibrous longitudinal muscle of the rectum at the apex of the perianal space and continued medially through the center of the hemorrhoidal pedicle at its proximal attachment to emerge through the rectal mucosa. (Fig. 4.) The needle is cut away and each of the remaining sutures is tied tightly on its respective side of the point of transfixion. Advantages of this method of high ligation are first, perfect hemostasis assured by the inclusion of the fibrous longitudinal muscle of the rectum thereby anchoring the ligature and preventing slipping. Secondly the sensory nerves of the anus and the subcutaneous external sphincter muscle are distal to the point of ligation. These sensitive tissues are thus excluded from the ligature, thereby avoiding excess pain and sphincter spasm. After final inspection, the hemorrhoidal mass is excised 0.5 cm. distal to the ligature. The stump is replaced in the rectum to slough off spontaneously in about eight days leaving a smooth, cleanly granulating base.

The two remaining primary hemorrhoids are similarly treated as are secondary hemorrhoids when present. Other lesions such as mucosal fissures, hypertrophied papillas and infected crypts are excised independently or as part of the hemorrhoidal mass. After completion of a properly performed operation, a maximum of rectal mucosa remains intact between excised areas, thereby insuring freedom from stricture.

The anorectal region cannot be adequately sterilized nor can it remain

surgically clean after operation. To attempt primary closure of the skin wound is to invite disaster in the form of infection.

tion prevents pooling of the oil and improves its distribution.

Retraction sutures are now removed, a

External hemorrhoid

Lateral mucosal incisions at hemorrhoidal pedicle (1 and 2)

Internal hemorrhoid
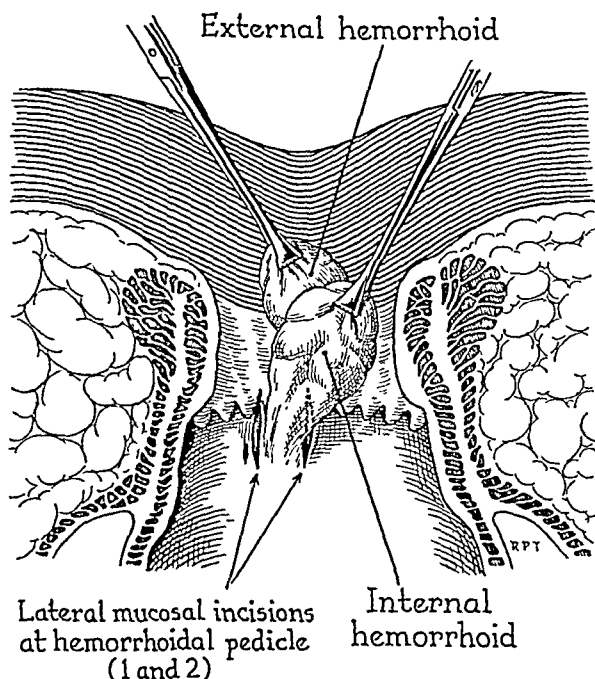
FIG. 2. Incisions at the base of the hemorrhoidal pedicle.

Consequently, to promote better dependent drainage, skin defects are left open to heal by granulation easily and painlessly in two to three weeks.

In hypersensitive patients, postoperative comfort may be further enhanced through the use of anesthetics, eucupin, anucaine, butecain or numerous others, in oil. When injected into the sphincter muscles, partial paralysis ensues which results in freedom from spasm for one to three weeks thereby increasing postoperative comfort. These chemicals are tissue irritants as shown by Kilbourne[11] and in patients whose tissue vitality is low, diabetics, nephritics and cardiacs, the use of chemicals is contraindicated because of the likelihood of slough formation. When utilized, 10 cc. of the warmed oil is injected through the denuded wounds into the external and internal sphincter muscles, these being supported during injection, on the crooked index finger placed within the rectum. Gentle massage of the muscle after injec-

thin sliver of vaseline gauze is inserted in the anal canal to separate raw surfaces, gauze dressings are applied and held in place by wide adhesive straps across the buttocks. "T" binders immediately postoperatively are unsatisfactory as they require constant adjustment to maintain dressings in place.

POSTOPERATIVE REGIMEN

Before the patient is removed from the operating room, 30 cc. of 0.5 per cent aqueous solution of mercurochrome is instilled into the urinary bladder per catheter. This procedure has resulted in reducing to less than 1 per cent the number of patients with postoperative urinary retention requiring catheterization (Woodruff and TeLinde),[12] (Helfert and Granet).[13]

Directly postoperatively, the patient is allowed a liberal high vitamin, low roughage diet; sedation for pain is prescribed as required; heat is applied to the perineum and the patient is allowed out of bed to

void if necessary. Twenty-four hours after operation the dressing and vaseline gauze are removed and sitz baths, two or more
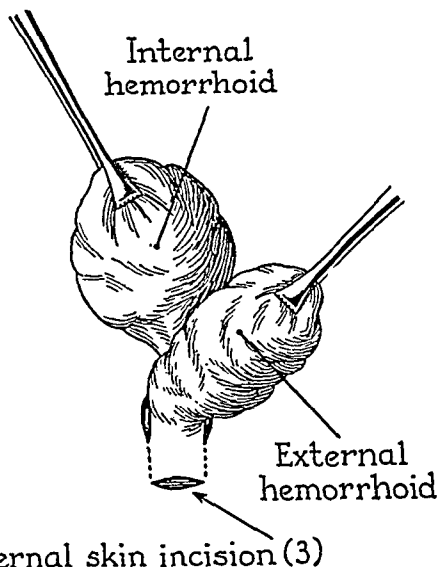
FIG. 3. External skin incision.

daily, are administered. The patient is allowed up and one ounce of petroleum-agar emulsion is administered daily. Usually, defecation takes place on the third day or if delayed, is stimulated by a small warm water enema. The patient can be discharged from the hospital on the fourth or fifth day. He continues on petroleum-agar and sitz baths and after defecation is required to take a warm rectal lavage, self administered, through a No. 24F urethral catheter. This procedure cleanses the granulating surfaces of residual feces and improves the comfort of the patient. After three weeks, epithelialization of the wounds should be complete. After unusually extensive operations, physiologic swelling of scar tissue may occur at about the sixth week which may narrow the lumen of the anal canal to admit only the fifth finger when palpated. Dilatation at this time is contra-indicated and unnecessary inasmuch as swelling soon subsides and the anal canal regains normal width.

CONCLUSIONS

1. Varices of the hemorrhoidal plexus (hemorrhoids) cause symptoms when com-

plicated by lesions resulting from trauma, infection or prolapse.

2. Sclerotherapy is limited in its applica-
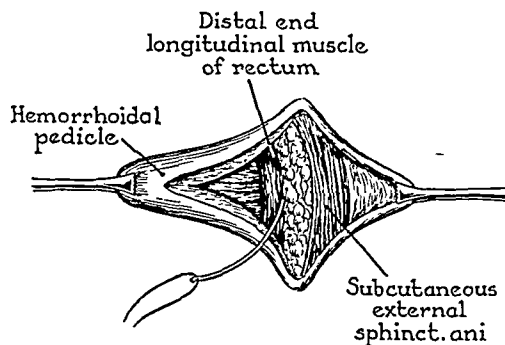
FIG. 4. Dissection completed showing the point of transfixion of the hemorrhoidal pedicle.

tion and is successful only in patients with small uncomplicated internal hemorrhoids. It is useful in the treatment of hemorrhoidal bleeding in patients who are severe cardiacs, nephritics and in the senile. When utilized, the patient must be warned of its temporary efficacy and of the high recurrence rate.

3. Hemorrhoidectomy when properly and accurately performed implies erradication of all hemorrhoidal varicosities. Most so-called "recurrences" result from physiologic enlargement of secondary varices remaining after an incomplete hemorrhoidectomy. This may result from a hurried, poorly planned or careless operation or from the timidity of an inexperienced surgeon who rightly fears stricture formation.

4. Patients complaining of rectal bleeding *must* have a carefully performed sigmoidoscopic examination prior to hemorrhoidectomy. By so doing, we can avoid the tragedy of discovering advanced rectal cancer in patients recently operated upon for "bleeding piles."

5. The operative technic of the modified "ligature" operation is described. Features emphasized are: (a) Caudal epidural nerve block anesthesia; (b) use of the Hullsieck anal retractor and the jack-knife position; (c) minimal denudation of the mucosa of the rectum and the mucoderm of the anus; (d) absolute hemostasis by the use of a

nonslipping transfixion ligature; (e) exclusion of the subcutaneous external sphincter muscle and the sensory nerves of the anus from ligation, thereby minimizing pain, edema and spasm; and (f) when not contraindicated, the use of oil soluble anesthetics to allay postoperative pain and spasm in hypersensitive patients.

6. Postoperative measures designed to minimize debility and excess nursing care are: (a) Prevention of postoperative urinary retention by instillation of 0.5 per cent mercurochrome solution in the urinary bladder; (b) establishment of an ambulatory regimen including hot sitz baths after twenty-four hours; and (c) immediate administration of a low roughage but full caloric and high vitamin diet.

REFERENCES

1. Buie, L. A., Bargen, J. A. and Rankin, F. W. The Colon, Rectum and Anus, p. 635. Philadelphia, 1932, W. B. Saunders.

2. Gabriel, W. B. The Principles and Practice of Rectal Surgery P. 40. London, 1932. H. K. Lewis.

3. Hirschman, L. Synopsis of Ano-Rectal Diseases. P. 205. St. Louis, 1937. C. V. Mosby.

4. Swinton, N. W. The injection treatment of hemorrhoids. *Labey Clinic Bull.*, 1: 21, 1940.

5. Rosser, C. Chemical rectal stricture. *J. A. M. A.*, 96: 1762, 1931.

6. Levy, E. Anorectal musculature. *Am. J. Surg.*, 34: 141, 1936.

7. Milligan, E. T. C., Morgan, C. N., Jones, L. E. and Officer, R. Surgical anatomy of the anal canal, and the operative treatment of hemorrhoids. *Lancet*, 2: 1119, 1937.

8. Jennings, W. B. and Karabin, J. E. Incidence of headache, nausea and vomiting following spinal anesthesia. *Am. J. Surg.*, 46: 317, 1939.

9. Labat, G. Regional Anesthesia. P. 444. Philadelphia, 1922. W. B. Saunders.

10. Hullsiek, H. E. New anal retractor. *Minnesota Med.*, 20: 300, 1937.

11. Kilbourne, N. J. Local anesthetics producing prolonged analgesia. *Surg., Gynec. & Obst.*, 62: 590, 1936.

12. Woodruff, J. D. and Te Linde, R. W. Postoperative care of the urinary bladder. *J. A. M. A.*, 113: 1451, 1939.

13. Helfert, I. and Granet, E. The prevention of acute urinary retention following anorectal surgical proceedures. *Am. J. Surg.*, 53: 129, 1941.

CLASSIFICATION OF WOUNDS AND THEIR TREATMENT*

B. BERNARD KAYE, M.D.   AND   WILLIAM M. ENNIS, M.D.

Instructor in Traumatic Surgery, Columbia University   Consultant Surgeon, Kings County Hospital

BROOKLYN, NEW YORK

THE present military conflict plus the mobilization of industry is clearly proving the importance of early adequate prophylactic treatment of traumatic wounds. A sound knowledge of the practical principles of surgery of wounds is essential if we are to minimize the military and industrial disabilities.

The prevention of contaminated wounds from progressing to the infected stage may well have a definite bearing on the outcome of this war. The reduction in the morbidity of wounds in our industrial plants and on the battle fronts will eliminate the loss of manpower so necessary in this all out struggle. First aid treatment should reduce the possibility of further contamination also the avoidance of additional trauma to that already present. Bleeding should be controlled by a pressure pad over the wound or the proximal artery. A tourniquet may be necessary. Packing of the wound is to be avoided if possible. The wound should be covered with a sterile dressing. Rapid evaluation of loss of the physiological function of the part involved may be attempted if circumstances permit. Splinting should be done and rapid transportation to the proper facilities made available so that immediate treatment can be instituted.

All traumatic wounds are contaminated and potentially infected. The source of contamination in traumatic wounds is fourfold, namely, the penetrating instrument, the surrounding soil, the patient's skin and the bacteria of the first aid work or physician. Therefore, meticulous asepsis should be maintained with the use of masks and sterile gloves. Clinical and experimental evidence-show that there is no method in use which renders a contaminated wound absolutely free of bacteria.[2] Therefore, the question as to whether a given wound remains aseptic or becomes infected, depends upon the virulence and type of bacteria which remain in the tissues, local conditions of moisture and temperature, and the resistance of the host.

The principles of treatment of traumatic wounds has been mentioned repeatedly by Mason,[9] Koch,[6] Kreuscher,[7] Reid and Carter.[10]

We shall discuss different types of traumatic wounds and their treatment:

1. *Abraded.* Simple cleansing should be done followed by the application of sterile or antiseptic ointment.

2. *Incised. Contaminated:* These are usually caused by stab wounds, glass, knives or other sharp instruments. The skin in the field of operation is shaved and scrubbed with tincture of green soap up to the edges of the wound. Grease and dirt are removed with benzine or ether. The skin is cleansed with iodine and alcohol, severed nerves and tendons are repaired and primary suture of skin is effected.

Infected. The wound is packed with moist saline gauze for forty-eight hours. Sulfathiazole powder may be added locally. Healing occurs by secondary intent.

3. *Puncture.* Cauterization of the tract with evacuation of the underlying hematomas, when present, is carried out.

4. *Lacerated. Contaminated:* Multiple superficial wounds due to peppering with tiny fragments of low velocity, as hand grenades and buck shot, are a combination of shallow puncture and lacerated types and may not require surgery.

Perforating wounds with small entrance and exit openings are usually the result of long range rifle bullets, sharpened or high velocity bullets. Bullets may traverse bone

and joint leaving a simple tunnel with no foreign bodies, clothing or débris remaining in the wound. Simple immobilization of the limb in plaster may be sufficient treatment.

Penetrating and perforating wounds require mechanical cleansing. Treatment of these wounds consists in thorough mechanical cleansing under anesthesia, general or regional, without further traumatizing or contaminating the field. Infiltration anesthesia should never be attempted, since it means infiltration of dirty, infected and traumatized tissue, with further devitalization of the part.

A blood pressure cuff inflated to about 200 to 250 mm. of mercury will control bleeding of the upper extremity and enable proper visualization. The tourniquet should be applied for the lower extremity with just enough pressure to shut off arterial pulsations.

A dry shave should be given in hairy sections. The surrounding tissues should be carefully cleansed with a solvent and thoroughly washed with sterile water. The wound is covered with sterile gauze. Antiseptics are never poured into the wound, because of the creation of nonviable tissues which afford excellent culture medium for bacteria. Antiseptics may destroy some organisms but devitalize the tissues and therefore seriously impede the inflammatory response of damaged tissue which is so necessary in repair, and also impair the effectiveness of the natural defense mechanism of the soft tissues. Normal saline solution does not damage soft tissues and it allows tissue to vitalize defense against remaining organisms. Chemical antiseptics cause interference with repair of bone, presumably the result of devitalization of the osteogenic elements. The skin may be painted with an antiseptic up to the wound margins. The wound itself is then irrigated with copious quantities of normal saline.

Débridement. Drapes are changed and débridement is performed. Adequate exposure by incising skin in the longitudinal axis of the limb may be necessary. Useful instruments are a large scalpel and a large tissue forceps with multiple teeth. Crushed and devitalized margins of skin flaps are excised. A quarter of an inch margin of skin around the wound is ample. Avoid unnecessary transverse division of uninjured skin and muscle. All contaminated and nonviable tissues should be removed. All torn and devitalized bits of fat, muscle and fascia are excised. Remove the contents and walls of the track in penetrating wounds, as the wound track is lined by devitalized and necrotic tissues. This requires sharp dissection and the avoiding of injury to important nerves and vessels. Tubular excision of perforating wounds of the tunnel variety is carried out. Completely detached fragments of bone are to be removed, avoiding bone fragments which remain attached to the periosteum, especially if they are essential in providing continuity of bone tissue. Excision in mass of all torn, crushed, discolored and noncontractile muscle will be encountered. The excision may involve sacrifice of all that remains of a muscle or maybe a muscle group. Massive necrosis and infection will supervene unless muscles with compromised blood supply are removed. Open up the adjacent cellular spaces affected by the trauma and when necessary incise the soft tissues, following up the cellular spaces in the depths of the wound, always keeping in mind the need for adequate drainage. When deep cavities are present, drainage must be insured by opening up the aponeurotic planes and the intermuscular spaces. Thorough débridement will convert a potentially infected accidental laceration into a nontraumatized aseptic wound. (Facial wounds permit limited débridement.)

The removal of foreign bodies includes war missiles, soil, soiled pieces of clothing and detached bony fragments. The wound is to be thoroughly irrigated with a saline solution; many liters should be used. The wound should be retracted to expose its entire extent and depth.

Appraisal and Repair of Anatomical Structure. An inspection is made of the

wound with appraisal of the underlying anatomical structures and their altered physiological function. An accurate plan is determined for the immediate or subsequent treatment of the injury. In war wounds the repair of damaged tissues, repair of divided capsules and the suturing of divided nerves and tendons are consigned to a later time. Tendons and nerves can be tagged with black silk for future reference. Fractures are to be reduced.

Release of Tourniquet. After the tourniquet is released the bleeding vessels are ligated.

Chemotherapy. Implantation of sulfa drugs directly into traumatic wounds has lowered the incidence of infection.[3,4,5,11] Sulfonamides are very effective bacteriostatic agents and when applied to tissues cause no apparent damage to the cells and do not alter the normal course of repair of either soft tissue or bone; as a matter of fact, they aid repair by preventing infection. Five to 10 Gm. of sulfanilamide or sulfathiazole powder is sprinkled in the wound as a prophylactic measure. One Gm. is allowed for every ten square inches of wound surface. Avoid massive aggregations of the powder at one point. Administer sulfonamides orally—4 to 6 Gm.—followed by successive doses of 1 Gm. every four hours, or subcutaneously 10 Gm. of sulfanilamide powder dissolved in 1,000 cc. normal saline, at six-hour intervals.

Fresh wounds are closed in layers in civil practice. Primary suture is never used in lacerated gunshot wounds.[12] Tangential wounds with loss of covering tissue are skin grafted primarily, in civil practice. Gaping tension wounds are packed with vaseline gauze. Oozing may be controlled with a pressure sponge from without. War wounds call for open packing plus immobilization in position of optimum function. Packing gives support to the tissues from within, preventing edema, and replaces bulk of tissue removed.

The Carrel-Dakin treatment is instituted when total wound excision has been impossible. It encourages wound drainage and will encourage separation of sloughing tissues. It is also useful for sterilizing the surface of a wound prior to skin grafting. The disadvantage is having the patient bedridden.

Splinting and Elevation of Part. All wounds of the extremities are immobilized either in traction, splinting or casting—moulded plaster cast without padding. The closed plaster method provides rest for the extremity, prevents reinfection of the wound and is a dressing which allows pus and secretion to seep away without disturbing the granulation tissue. Indications for removal of plaster are pain, looseness and softening of the cast, secondary hemorrhage and foul smell.

Trueta[12] in his experience in the Spanish War claims the value of immobilization of a wounded limb in a complete plaster of paris cast: (1) Rest allows venous and capillary thrombi to form; (2) rest allows new capillaries to form which are not torn by repeated dressing of the wound; (3) plaster maintains a constant beneficial pressure on the wound; (4) the mixture of organisms on the wound may by their mutual antagonism prevent the victory of any one group; and (5) an uncovered deep wound produces dehydration and loss of heat.

Infected Wounds. The contaminated wound has gone on to the infected state. The bacteria from the soil, skin, clothing or intestine have multipled and have been destroyed by the forces mobilized by the inflammatory reaction. The character of the infection depends on:[8] number and virulence of the bacteria, the pathologic character of the inflammatory response to the host to the particular type of bacteria concerned, the blood supply of the contaminated part, and the amount of devitalized tissue in the area, immunity to the host, and the presence or absence of anatomic barriers which will confine the infection within a compartment.

Contaminated war wounds left untreated always go on to infection. Bacteria have multipled and invaded the lymphatics, and

the absorption of toxins results in clinical rise in pulse and temperature. Surgical procedures at this stage may become dangerous and produce spreading of the infection with severe general reaction.

TREATMENT

Chemotherapy is to be instituted depending upon the type of infection. Sulfanilamide is administered for streptococcal infections and sulfathiazole for staphylococcal and gas gangrene infections as follows: Initial dose 6 Gm. followed by 1 Gm. every four hours; this should be given every six hours for about one week. The open wound should be dusted with the sulfonamide powder.

Surgical therapy includes decompression of pus, excision of necrotic and dead tissue, and removal of foreign bodies.

Surgical Indications. These are localization with suppuration and fluctuation. In war wounds, there will be pain, swelling and tenderness along the wound track. Thin, foul, sanguineous discharge is usually present in deep anaerobic infections. A spreading mottled bronzing discoloration of the surrounding skin requires removal of dead muscle tissue and foreign bodies.

The Carrel-Dakin treatment of necrotic sloughing wounds in which surgery is contraindicated may be used. The removal of dead tissue will enhance the effectiveness of sulfonamide or zinc peroxide.

Compound Wounds. The soft tissues are treated in a similar manner to those surrounding simple lacerated wounds, namely, mechanical cleansing, saline irrigations plus débridement.

All traumatized muscles, fascia and skin edges are excised. Blood clots are evacuated. Loose, small bone fragments are removed and soiled bone surfaces are excised. Complete hemostasis is desired. Continuous irrigation with large quantities of saline solution is carried on throughout the procedure. Chemotherapy is applied locally, supplemented by oral doses of 5 to 8 mg. per 100 cc. for prophylactic effect.

Traction and manipulation are carried out to reduce the fracture; skeletal traction may be necessary. Internal fixation is used in some clinics.

Surgeons differ as to the best method of closing a wound, the common methods employed are: Immediate closure with no drainage is to be attempted only in civil practice. War wounds are never closed by primary suture. Primosecondary suture may be brought together after forty-eight to seventy-two hours.

Orr-Trueta Treatment. This is a combination of the Orr treatment of osteomyelitis and the Boehler treatment of fractures. Treatment and débridement is to be followed by reduction and retention of fracture, by means of a plaster of paris splint encased in circular plaster. Primary suture or wounds are packed with vaseline gauze, sulfanilamide paste gauze or 12 per cent lactose solution gauze. This prevents secondary introduction of pathogenic bacteria and also forms a barrier against spreading infection.

Carrel Dakin tubes are used with open wounds and are advisable in those cases in which wound excision has been incomplete. It is more valuable in sloughing wounds, as it encourages wound drainage; but this requires adequate facilities and observation.

Retention is best accompanied by fixation of all parts so that there is perfect control without constriction or pressure. This is usually accomplished by plaster of paris cast. Aftercare is essential to maintain such control and to protect against secondary infection.

SUMMARY

In conclusion we are offering an outline with a view to simplifying the approach to the treatment of traumatic wounds, which we believe will be helpful in civil and military practice.

1. Abraded
2. Incised—contaminated, infected
3. Puncture
4. Lacerated

A. Contaminated:
 (1) Multiple superficial wounds
 (2) Perforating (through and through)
 (3) Penetrating and perforating
  Mechanical cleansing
  Copious saline irrigation
  Débridement—skin, muscle, devitalized bone
  Removal of foreign bodies
  Saline irrigation
  Appraisal and repair of anatomical structure: nerves and tendons; fractures
  Release of tourniquet
  Chemotherapy
  Wound closure—primary, primary secondary, open with packing
  Splinting and elevation of part

B. Infected:
 Chemotherapy
 Surgical
 Chemical

5. Compound
 Saline irrigation
 Débridement
 Reduction of fracture

Wound closure
Fixation
After care

REFERENCES

1. BAILEY, HAMILTON. Surgery of Modern Warfare. Vol. 1, p. 67. Baltimore, 1941. William & Wilkins Co.
2. BISGARD and BAKER. Treatment of fresh traumatic and contaminated surgical wounds. *Surg., Gynec. & Obst.*, 74: 20, 1942.
3. CAMPBELL, W. C. and SMITH, H. Fresh compound fractures. Treatment by sulfonamides. *J. A. M. A.*, 117: 672, 1941.
4. JENSEN, N. K., JOHNSRUD, L. W. and NELSON, M. C. The local implantation of sulfanilamide in compound fractures. *Surgery*, 6: 1, 1939.
5. KEY, J. A., FRANKEL, C. S. and BURFORD, T. H. The local use of sulfanilamide in various tissues. *J. Bone & Joint Surg.*, 22: 952, 1940.
6. KOCH, SUMNER L. The primary treatment of wounds. *Minnesota Med.*, 24: 747, 1941.
7. KREUSCHER, PHILIP, H. The treatment of fresh wounds. *Am. J. Surg.*, 51: 573, 1941.
8. LOCKWOOD, J. S. *Surg. Clin. Nortn America*, 21: 1739, 1941.
9. MASON, MICHAEL S. Wound healing. *Illinois M. J.*, 78: 523, 1940.
10. REID, M. R. and CARTER, B. N. The treatment of fresh traumatic wounds. *Ann. Surg.*, 14: 4, 1941.
11. TENDLER, M. J. Sulfanilamide in surgery. *J. Tennessee M. A.*, 33: 300, 1940.
12. TRUETA, J. Treatment of War Wounds and Fractures. P. 22. New York, 1940. Paul B. Hoeber.

DIFFERENTIAL DIAGNOSIS BETWEEN THYMIC DUCT FISTULAS AND BRANCHIAL CLEFT FISTULAS*

REPORT OF A CASE OF BILATERAL AURAL FISTULAS AND BILATERAL THYMIC DUCT FISTULAS

CONRAD J. BAUMGARTNER, M.D. AND S. STEINDEL, M.D.

LOS ANGELES, CALIFORNIA

SEVERAL types of cysts and fistulas are encountered in the neck. These are the well known midline lesions such as thyroglossal, dermoid, and the lateral cysts and fistulas of branchiogenic origin, and the hygromas.

Although many cases of congenital lateral cervical fistulas and auricular fistulas have been reported in the literature, it is rare to find a case which presents the picture of both bilateral thymic duct fistulas and bilateral branchial fistulas. Hyndman and Light,[1] in 1929, reported a case of a colored boy, age four, who illustrated this rare finding. This paper presents a similar case. In addition, the case illustrates that lateral cervical fistulas may be the remnants of true thymic duct fistulas and gives us the opportunity to differentiate those lateral cervical fistulas and cysts that have their origin in true pharyngothymic duct from those whose origin is the branchial clefts, and/or pouches.

CASE REPORT

A negress, R. S., age twelve, was first seen in the out-patient department of the Los Angeles County General Hospital, complaining of bilateral draining sinuses in the neck and at the ears. All four openings had been draining periodically since birth. She had bilateral fistulous openings along the anterior border of the sternomastoid muscles at the middle third, and over the anterior border of the ascending limb of the helix of both ears. (Figs. 1 and 2.) The findings otherwise were negative, except for a very low mentality and difficulty in talking.

A diagnosis was made (1) of bilateral true congenital auricular fistulas, coming from the first branchial groove and, (2) bilateral pharyngothymic duct fistulas, originating from the third branchial pouch.

On December 30, 1941, under anesthesia, the lateral neck tracts were excised after the tract had been injected with methyl blue. The incisions were made along the course of the anterior border of the sternomastoid muscles, first on the right and then the left. On the right side, the tract ended abruptly at a level of the hyoid bone. On the opposite side, it passed in the classical manner, under the anterior portion of the sternomastoid, over the jugular vein and under the facial vein. At the middle portion of the posterior belly of the digastric muscle it arched medially, ending in the pharynx just below the tonsil. After dissection was carried beyond the digastric muscle, all but a stump of the tract was removed and a probe passed into the lumen, suturing the same securely and then pulling the probe and tract into the pharynx where the then inverted tract was ligated intra-orally. The wounds healed *per primum*.

The excision of the auricular fistulas was performed on January 13, 1941. Both tracts followed along the external auditory canal to the vicinity of the tympannic membrane, where both ceased. The postoperative course was uneventful, except for a short period of purulent drainage from the right ear. The child was again seen December 5, 1941, at which time keloids had formed at the scars, but there had been no recurrence of drainage.

The pathologist's findings were as follows: (1) The lateral neck fistulas (Fig. 3): Microscopic sections show the lumen containing desquamated epithelium and collections of cells, mostly lymphocytes. The lumen is lined by respiratory-like epithelium. (2) The auric-

ular fistulas (Fig. 4): Microscopic sections show hair follicles, stratified squamous epithelium with keratinization and papillae. The lumen

an internal opening into the pharynx, or an anterior opening externally on the anterior border of the sternomastoid; or there may

FIG 1 View showing fistulous opening along the middle third of the anterior border of the right sternomastoid, and over the anterior border of the ascending limb of the helix of the right ear.

FIG. 2. View showing fistulous opening along the middle third of the anterior border of the left sternomastoid, and over the anterior border of the ascending limb of the helix of the left ear.

contains desquamating cells and cornified layer. Sebaceous glands are also seen.

CLINICOPATHOLOGIC CORRELATION

The clinical manifestations of lateral cervical cysts and fistulas are varied and may be seen at any age or in any race. Hyndman and Light (1929), in a collection of ninety cases, found that the average age at which these patients came to the doctor is twenty-one. The range varies from three weeks to forty-eight years. Usually, the cyst is first noticed when it reaches the size of a small egg. In the ninety cases, 84 per cent of the total were unilateral; 52 per cent being on the right and 32 per cent on the left. The remainder were bilateral. The cyst may be painless but fluctuant, and the patient may give a history of variation in size in previous years. The cyst is usually located in the anterior triangle of the neck; but although it may be at any level, it is usually opposite the middle third of the sternomastoid. The cyst may have no opening, or it may become a fistula with either

be both internal and external openings. The patient may have noticed discharge, often of a sticky mucoid consistency, either into the pharynx or onto the skin through the external opening.

As to the origin of lateral cervical cysts and fistulas, there has been much discussion and disagreement. Wenglowski,[2] who probably did more exhaustive work in tracing the pathogenesis of these lateral lesions than any other person, shows that early in the development of the embryo there is a lateral out-pocketing from each of the third branchial pouches. Each of these tubular-like structures descends into the mediastinum and there takes part in the development of the thymus gland. The epithelium lining the duct is ciliated and resembles respiratory epithelium. But as the duct passes downward, its lateral wall comes in close contact with the stratified squamous epithelium of the cervical sinus. The cervical sinus is the result of the rapid overgrowth of the first and second branchial arches in such a manner that they

overlap and override the remaining branchial arches, and so form an overhanging roof to the remaining branchail clefts, much

lateral cervical fistulas. As further development takes place, islands of lymphocytes appear which invade and surround the duct
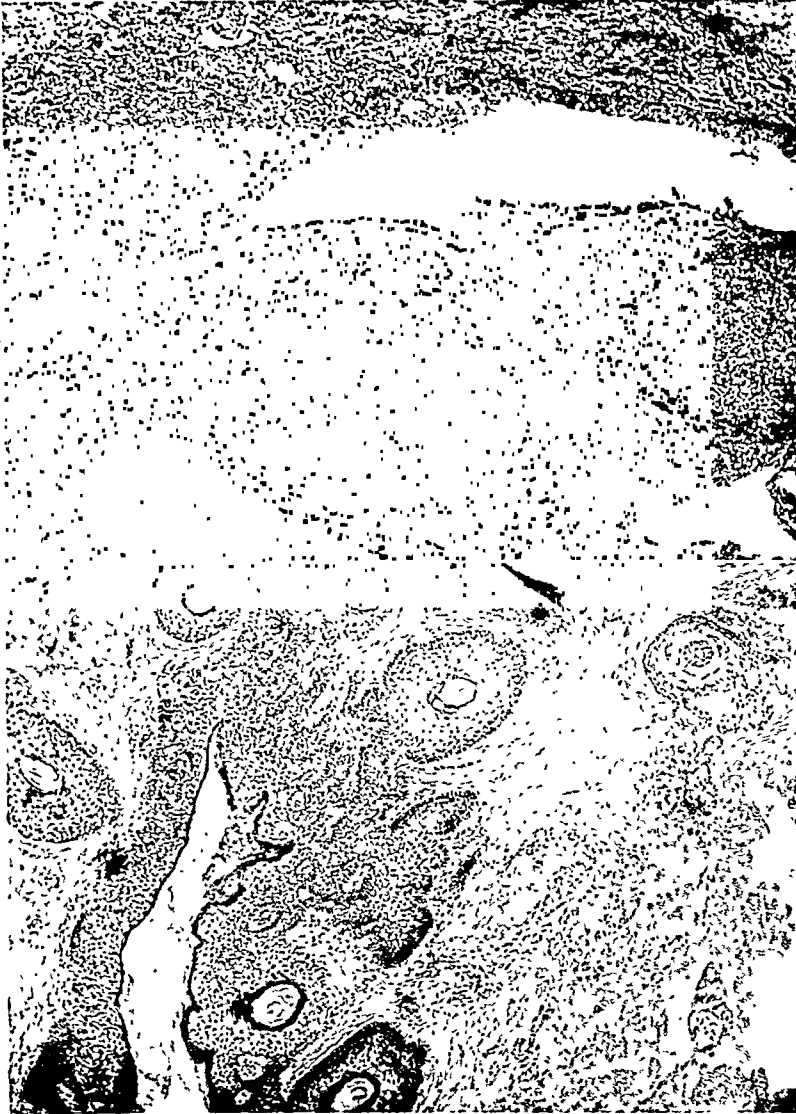
FIG. 3.

FIG. 4.

FIG. 3. Microscopic section of lateral neck fistula, showing respiratory-like epithelium and collections of lymphocytes, but showing no vestiges of skin.

FIG. 4. Microscopic section of the auricular fistula, showing hair follicles, stratified squamous epithelium, and sebaceous glands, characteristic of true branchial groove fistulas.

like the operculum in fish. The mesochymal wall between the cervical sinus and the thymic duct becomes very thin and the squamous epithelium lining the cervical sinus becomes part of the wall of the thymic duct. Because of this, both squamous and ciliated epithelium are found in certain

and form the anlage of the thymus gland. In microscopic sections of remaining branchial clefts, both areas of packed lymphocytes and follicles are found, and also, but rarely seen, are true Hassall's corpuscles. This observation has been offered as additional proof that so-called

branchial fistulas are actually the remains of the thymic duct.

The course of the fistula is also a clue to its possible origin. The course of the thymopharyngeal duct, because of its origin from the third branchial pouch, must be anterior to the carotid sheath and must have its origin in the infratonsillar fossa. In most cases of lateral fistulas, just this relationship exists. A fistula that has its origin in the second branchial pouch or cleft, would have to follow other courses, and would have to pass between the internal and external carotids, as pointed out by Malcolm and Benson.[3] Furthermore, a fistula having its origin solely in a branchial groove would contain vestiges of skin and according to Meyer[4] would have to be above the level of the hyoid bone.

Those who differ with Wenglowski, point out that this lateral cervical lesion may be the remains of either branchial groove, or branchial pouch and not necessarily be a remnant of the thymic duct. Fistulas have been found that may very well come from the second branchial pouch; having origin in the supratonsillar fossa and instead of being ventral to the carotid sheath, pass between the internal and external carotids. To explain both the presence of ciliated and stratified epithelium, it is pointed out that a lateral cervical fistula may be made up of a persistent branchial cleft and corresponding branchial pouch with perforation of the closing membrane between them. The ciliated pseudorespiratory epithelium comes from the branchial pouch, and the stratified squamous epithelium is derived from the cleft. The presence of packed bands of lymphocytes and even lymphocytic follicles may be due to the almost universal infection of these fistulas.

Lastly, some have stated that lateral anomalies may be due to a persistence of the cervical sinus with or without connection with the branchial apparatus.

The possibility of the fourth branchial apparatus playing a part in the development of congenital lateral cervical fistulas, may be dismissed by pointing out that since the arch of the aorta comes from the fourth arch, the fistula if derived from this arch, would have to course below the subclavian vessels on the right, and arch around the arch of the aorta on the left. There has been no such case reported, as far as we know, in which such a course has been described.

The auricular fistulas are more common than lateral cervical fistulas and may be present all during life without being noticed, except for a possibly small, dimplelike depression located on almost any part of the ear. The fistula or cyst may become infected and drain offensive discharge. If the secretions are retained, the cyst may become enlarged and tender, and at times, the infection may extend forward and an abscess be formed a short distance away from the fistulous opening. The anterior auricular lymph-node may become involved, swollen and tender. Once these cysts are infected, the tendency for remissions and recurrence of the infection is great.

As to the origin of the not uncommon auricular cysts or fistulas, there is very little disagreement. In general, two theories have been advanced. The first state that they are the result of malformation of the first branchial cleft. The second points out that early in the life of the embryo, six small nodules appear derived from the first and second branchial arches and surround the first branchial cleft. These rounded nubbins give rise to the various parts of the pinna. In the process of fusion of these nubbins, cysts or fistulas may develop. These are all derived from ectoderm and are lined with normal skin, including all the accessory structures such as hair follicles, sebaceous and sweat glands. (Fig. 4.) Studies of reconstructed models of various aged embryos seem to bear out the latter explanation.

The surgical technic for treatment of lateral cervical fistulas has been reviewed in a previous paper published by one of the authors (C. J. B.) in 1933.[5,6]

SUMMARY

The case herewith presented represents both the true branchial fistula, which affords an opportunity for clarification of a still greatly confused differential diagnostic problem.

For purposes of clarification, those lateral neck fistulas occurring below the hyoid in a definite relations to the sterno-mastoid and the carotid sheath, should almost without exception be called pharyngothymic duct fistulas and cysts, coming from the third pouch, rather than branchial cysts and fistulas. Those above the hyoid may or may not be of thymic duct origin.

True branchial cleft affairs are found in the area above the hyoid and should contain vestiges of skin such as sebaceous glands, sweat glands and hair follicles. Most of these originate from the first groove and terminate somewhere in the vicinity of the tympannic membrane.

Branchial fistulas do rarely originate from the second pouch, and would have to descend between the internal and external carotid arteries.

REFERENCES

1. HYNDMAN, OLAN R. and LIGHT, GEORGE. The branchial apparatus. *Arch. Surg.*, 19: 410–452, 1929.
2. WENGLOWSKI, R. Ueber den Halsfisteln und Cysten. *Arch. f. klin. Chir.*, 98: 151–208, 1912.
3. MALCOLM, R. B. and BENSON, E. R. Branchial cysts. *Surgery*, 7: 187–203, 1940.
4. MEYER, HERBERT WILLY. Congenital cysts and fistulae of the neck. *Ann. Surg.*, 95: 1–26, 1932. Congenital cysts and fistulae of the neck. *Ann. Surg.*, 95: 226–248, 1932. Footnote 3. True branchiogenic cyst and fistula of the neck. *Arch. Surg.*, 35: 766, 1937.
5. BAUMGARTNER, CONRAD J. Branchial and thyroglossal duct cysts and fistulae in children. *Surg., Gynec. & Obst.*, 46: 948–955, 1933.
6. BAUMGARTNER, CONRAD J. Lesions of the neck. *J. A. M. A.*, 113: 1860–1864, 1939.

HUMAN RED CELL CONCENTRATE FOR SURGICAL DRESSINGS*

JOHN J. MOORHEAD, M.D. AND LESTER J. UNGER, M.D.

NEW YORK, NEW YORK

IN the processing of blood plasma it is well known that over half of the whole blood withdrawn is discarded. It occurred to one of the authors (J. J. M.) that this red cell discard might serve a useful purpose as a wound dressing for raw and potentially infected surfaces, and the first clinical usage of the product on April 28, 1942 was at the New York Post Graduate Hospital in the case of a knee joint arthrotomy torn apart by a delirious patient. The tendency for infection to develop in the knee joint is well known, as is the unfavorable reaction of the synovial lining to antiseptics. In this case, the incision was approximately nine inches in length and the interior of the joint was fully exposed. Red cells of semiliquid consistency were placed in the joint until they overflowed the cavity. On alternate days the same procedure was repeated, and on each occasion, a massive gauze and cotton dressing was applied. After several applications, co-aptation with flamed adhesive was started and step by step the entire incision was thus closed. There was no unfavorable reaction locally or systemically, but instead the wound margins took on a healthy appearance and there were no excess granulations. This patient recovered with a movable knee.

The second case, extensive burns of both lower extremities, was a patient of Dr. Harry V. Spaulding under treatment at the Reconstruction Hospital Unit of the Post Graduate Hospital. There was a profuse purulent exudate and the patient's general condition was very poor. Treatment was undertaken by Dr. Spaulding based on the favorable outcome of the case just mentioned. The burned area of one thigh was cleansed with saline under intravenous anesthesia, and then the red cells were liberally applied to the entire test area so that the coverage resembled a coating of red paint. Response was so prompt and satisfactory that the same procedure was later adopted for the opposite thigh. The purulent exudate rapidly subsided under the painless film-like protective. Granulations were smooth and healthy and the patient's general state markedly improved so that massive grafts were later successfully applied. The outcome in this patient was outstanding, notably because many other types of treatment had been tried in vain.

In addition to these two cases, several others of lesser importance have been treated by our confreres at the Post Graduate Hospital and elsewhere.

In the group, are postoperative infections, and two resistant ulcers of the leg. The striking features appear to be the following: (1) Subsidence of purulent secretion; (2) smooth healthy granulations; (3) impervious veneer coverage preventing fluid loss and re-infection; (4) painless application, and (5) conversion of a waste product into a useful agency.

Burns, infected and non-infected wounds, and certain types of ulceration have been benefited in our limited experience. It has been used for an osteomyelitis of the carpus quite recently but the outcome cannot at this early date be stated.

This red blood cell concentrate was prepared from blood of healthy donors withdrawn under sterile precautions by a closed system in 5 per cent sodium citrate. This

was then centrifuged at 2,000 revolutions per minute for three-quarters of an hour and allowed to stand in a refrigerator at 3 to 5°c. After seven days the supernatant plasma was removed by a closed system. The cells of four individuals without regard for their blood groups were pooled and then returned to the refrigerator. Ten to twenty days elapsed from the time of the phlebotomy before the material was used. It was then quite viscid and had the consistency of thin red paint.

The total protein content was about 35 Gm. per 100 ml. with an albumin-globulin ratio of about 2:1. The hemoglobin content was 29 Gm. per 100 ml. The red blood cell count was approximately 8,000,000 per cu. mm. The leucocyte count 5,000 per cu. mm. with 1 per cent polymorphonuclear neutrophiles, 90 per cent lymphocytes and 9 per cent monocytes. Bacteriological examination showed the material to be sterile.

Many problems for further investigation immediately present themselves. Is it better to paint or spray the lesion and allow the material to dry into a crust or to use it as a wet dressing? Is it better to prevent evaporation and keep the dressing continuously moist or to allow it to dry? Is it better to use the material after actually hemolyzing all the cells or to omit this process? Is it better to use it as a fluid or to dry the material and use it as a powder? Is there any advantage in using cells of the patient's own blood group? It is too early definitely to answer these questions. These and others are, however, under investigation, including the addition of sulfa drugs and other antiseptics.

Several theories to explain the results obviously present themselves. Perhaps it acts as an occlusive and reduces oozing and re-infection. The very high protein content of the material may play a rôle. There may be some specific element which in this concentrated form is of value; or the products of destroyed and disintegrated cells may stimulate cell reproduction. Certainly leucocytes as living functioning phagocytes have disappeared, and likewise plasma with its contained antibodies are absent.

It is recognized that the number of patients treated is small; but because of our satisfactory experiences this preliminary report is presented with the hope that others will be stimulated to use this mode of therapy in an attempt to evaluate it. If these initial successes are substantiated, this hitherto discarded product will be of considerable value in treating war and other casualties.

BENIGN TUMORS OF THE MALE BREAST

MAJOR BUFORD WORD, M.C.   AND   LT. COL. WILLIAM C. REED, M.C.
Assistant Chief of Surgical Service, Station Hospital,   Chief of Surgical Service, Station Hospital, U. S. Army
U. S. Army

CAMP SHELBY, MISSISSIPPI

TUMORS of the male breast are un- common, comprising only approxi- mately 2 per cent of all neoplasms affecting the breast. Due to the relative rarity of the lesion as compared with tumors of the female breast, reports on the subject are so infrequent that we have been prompted to describe the cases seen at this hospital since its organization. The pathological and therapeutic significance of lesions of the male breast become real and not apparent where the patient-popu- lation of a large hospital is exclusively male.

During the seventeen-month period from October 18, 1940, to March 26, 1942, there were 29,128 admissions to the Station Hospital, Camp Shelby, Mississippi. Of this number twenty were admitted for complaints referable to the breast, an incidence of one breast lesion in each 1,456 admissions. All of the lesions were benign and all occurred in white patients except one (Indian); three were inflammatory in character and seventeen were abnormal growths. One neoplasm, one cystic dilata- tion of a duct, and fifteen cases of simple hypertrophy composed the non-inflam- matory group of cases. De Cholnoky,[1] in reporting a series of 102 benign tumors of the male breast, found thirty-five inflam- matory lesions, thirty-six fibromas or fibro-adenomas, three gynecomastias and one lipoma. No cystic lesions were noted in his series. The inflammatory lesions were all abscesses and are tabulated in Table 1. In the non-inflammatory group of cases the three different pathological en-

tities encountered, namely, lipoma, cystic dilatation of a duct and gynecomastia, will be discussed in detail.

Table 1 lists in concise form the breast lesions seen at this hospital since its organization.

Finney, as quoted by Guy,[2] states that pure lipoma of the breast proper is one of the rarest tumors regardless of age. White,[3] in 1927, described a lipoma of the left breast that occurred in a white male car- penter, age sixty-four. A history of forty years' duration of the tumor was obtained from the patient. Guy also reported a case of lipoma in a white male, age four and one-half years. However, White's case is the only one found in a brief search of the literature similar to the case herein reported.

CASE REPORT

Pvt. S. J. B., age twenty-five, Hospital Register No. 11,842 (Case No. 3, Table 1), was admitted to Station Hospital, Camp Shelby, Mississippi, May 31, 1941, with the chief complaint of tumor of the left breast. The soldier stated that in 1922, at the age of six, he fell and bruised his left breast. Soon after the accident he noticed a swelling in this region. The left breast gradually enlarged from the onset until the present time. The patient had never noticed any discharge from the nipple and only occasionally had he noticed soreness in the left breast. There was no complaint other than the one described above. The soldier's family history, to his knowledge, was negative for a similar condition in any of the male members.

The physical examination revealed a well developed and well nourished white male with no abnormal findings except for an apparently normal female breast on the left side. Examination of the left breast revealed a large soft mass about the size of a grapefruit. No lumps or tender areas were noted and there was no

All laboratory tests, including blood serology, urinalysis, and x-ray of the chest were within the limits of normal. On June 12, 1941, left mastectomy was done under gas-oxygen-ether anesthesia. The patient's postoperative convalescence was uneventful. The wound healed by primary union.

TABLE I

Case	Initials and Hospital Register Number	Age	Breast	Duration of Symptoms	Trauma	Operation	Diagnosis
1	S. T. L. 999	23	Left	10 days	Yes	Incision and drainage	Abscess
2	J. C. F. 8,636	18	Left	5 months	No	Excision of mass	Gynecomastia
3	S. J. B. 11,842	25	Left	19 years	Yes	Mastectomy	Lipoma
4	F. B. 13,242	21	Right	2 years	No	Mastectomy	Gynecomastia
5	G. F. H. 13,961	22	Left	6 years	Yes	Mastectomy	Gynecomastia
6	E. A. D. 14,837	34	Left	13 years	No	Mastectomy	Cystic dilatation of duct
7	D. Y. M. 15,035	19	Left	4 years	No	Mastectomy	Gynecomastia
8	J. C. R. 17,119	24	Right	3 months	Yes	Mastectomy	Gynecomastia
9	N. B. G. 18,121	22	Left	5 years	No	Mastectomy	Gynecomastia
10	W. P. S. 19,958	23	Right	6 months	No	Mastectomy	Gynecomastia
11	D. L. K. 20,143	25	Right	2 months	No	Mastectomy	Gynecomastia
12	J. D. M. 20,218	23	Right	1 year	No	Mastectomy	Gynecomastia
13	K. D. T. 20,302	24	Left	15 years "All his life"	No	Mastectomy	Gynecomastia
14	J. B. M. 21,028	24	Right	"Pain 2 years" Mass 3 months	No	Mastectomy	Gynecomastia
15	C. T. W. 23,147	22	Left	1 week	No	Incision and drainage	Abscess
16	J. C. F. 24,066	18	Left	13 months	No	Mastectomy	Gynecomastia
17	O. K. A. 25,640	27	Right	10 years	No	Mastectomy	Gynecomastia
18	R. H. F. 26,280	26	Right	1 week	Yes	Incision and drainage	Abscess
19	R. O. M. 27,020	32	Right	2 weeks	No	Mastectomy	Gynecomastia
20	J. J. S. 29,128	22	Right	3 years	No	Mastectomy	Gynecomastia

discharge from the nipple. The underlying structure did not appear to be attached to the chest wall or to the skin superficial to the mass. When the patient contracted the pectoral muscles of the left chest wall the tumor mass could be seen to divide into quadrants.

Pathological examination revealed the tumor to weigh 1,090 Gm. It was 23 cm. long, 15 cm. wide, and 10 cm. thick. It was bluntly triangular in shape and had a lobulated appearance. It was firm and well encapsulated. The capsule of the mass was somewhat mottled and

translucent. On cross section the parenchymatous tissue was of a lemon-yellow color. The cut surface was covered by a fine, dewdrop-
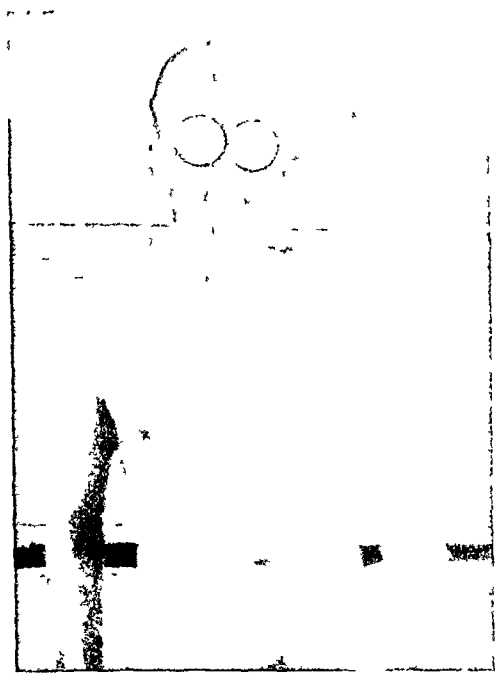

FIG. 1. Case III. It is noted in the above photograph that the patient is distinctly masculine except for left breast.

like deposit composed of minute fat droplets. There were strands, pink in color, and varying

of striped muscle bundles. Apparently the muscle bundles were separated from each other by the increasing deposit of fat tissue. There was no evidence of malignancy. Diagnosis: Lipoma, replacing voluntary muscles.

White's description of the clinical features of the lesion in his patient is strikingly similar to our case in one respect: "The skin is everywhere freely movable over the tumor and the tumor over the deeper structures, when the left pectoral muscle was tensed, the tumor rose up, stood forward and became less movable as if attached to the pectoral muscle"; in our case, "When the patient contracted the pectoral muscles of the left chest wall the tumor mass could be seen to divide into quadrants." This observation led us to suspect that the lesion was not simple hypertrophy but neoplastic, as the reaction of the tumor, when the left pectoral muscles were tensed, showed an abnormal attachment to the chest wall.

Cystic dilatation of a duct was found only once and should not be confused with cystic disease of the breast in the female which is known by almost as many names as there are authorities who have described

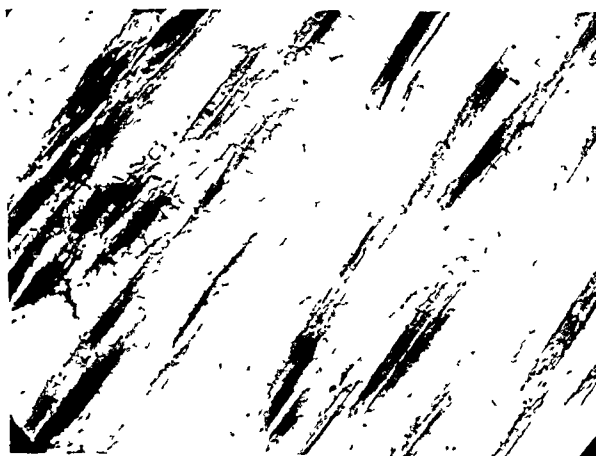FIG. 2. Photomicrograph of tissue from left breast, Case III, showing "fat cells" between striated muscle fibers (pectoralis major muscle).

in thickness and length, traversing the fat matrix. Microscopic: The bulk of the tissue was composed of fat cells. They surrounded strands

the condition. The lesion occurred in the left breast of an Indian patient, age thirty-four, who gave a history of thirteen

years' duration. There was no trauma incident to the onset of the growth, to the patient's knowledge.

According to Boyd,[4] the most common type of large cyst is what Bloodgood describes as "blue-dome cyst" and may be seen at operation after the subcutaneous tissue has been divided. In our patient, although the cyst was only one dilated duct, the "blue-dome" was quite visible through the skin beneath the nipple. On inspection, it appeared to be about the size of a half-dollar.

CASE REPORT

Pvt. E. A. D., Indian, age thirty-four, Hospital Register No. 14,837 (Case No. 6, Table 1), was admitted to Station Hospital July 31, 1941, with the chief complaint of enlargement of the left breast. The soldier stated that he first noticed a small lump in his left breast in 1928. The mass made its appearance without trauma and had gradually increased in size since onset. There had never been any discharge from the nipple and no pain was associated with the lesion.

The general physical examination was non-contributory except for a dark skin over entire body and enlargement of the left breast.

was very thin and there was a bluish discoloration present.

Laboratory tests completed, including x-ray

FIG. 3. Case VI. Gross appearance of cystic mass of left breast.

of chest, complete blood picture, urinalysis, and blood serology, were all found to be within the limits of normal. On August 7, 1941, left mastectomy was done. The breast was removed
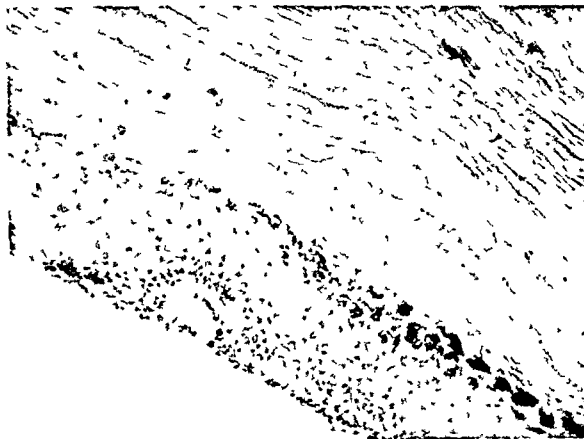
FIG. 4. Photomicrograph of cystic mass, Case VI, showing wall of the cyst proper. The section of skin containing pigment is not shown in the field.

On examination of the breast a mass about the size of a lemon was located just beneath the left nipple. The tumor appeared to be cystic to the palpating hand and was attached to the skin. The skin over the "fluid" or mass

by means of two transverse curvilinear incisions. The nipple was sacrificed because of the attachment of the tumor to the nipple and skin beneath the nipple. The operative recovery was uneventful and the wound healed by

primary union. The patient was discharged to duty on the twelfth postoperative day.

Pathological examination revealed the speci-
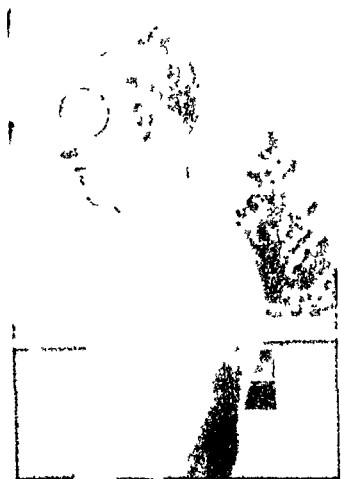
FIG. 5. Case VII. Simple hypertrophy of left breast.

men to consist of an elliptical section of white skin 8 by 5 cm. and the underlying mass of fibrofatty tissue, size 9 by 6 by 3 cm., 2 cm. from one end and 1 cm. from one border was the nipple. A cystic mass 4 cm. in diameter was seen and felt in the breast below and

end of the specimen merged with the cyst wall. At the other end a collection of fat was interbound between skin and the cyst. The cyst wall was composed of stratified squamous epithelium without skin appendages or rete pegs. The cells in its deeper layers were cornified and sloughing off. In the deeper layer of the cyst lining were masses of inflammatory cells. There was no evidence of malignancy. Diagnosis: Cystic dilatation of large duct of breast, left.

Gynecomastia is an enlargement of the male breast simulating in size and appearance a virgin female breast. The fundamental difference in structure between the male and female breast is that in the former there are no acini, but only a series of ducts of fair size. The pathologist tells us that the condition is simple hypertrophy of the tissues present and is not neoplastic. The microscopic appearance of gynecomastia is very similar to the microscopic appearance of fibromas and fibro-adenomas of the male breast. In fibromas there are no acini but fibrous tissue about the ducts is abundant, while in fibro-adenoma there

FIG. 6. Photomicrograph of breast tissue, Case VII, showing the ducts surrounded by dense layers of fibrous tissue.

medial to the nipple. This mass had a "bluish" color when viewed through the skin. On section the cyst was found to be unilocular and filled with flakes of caseous material and cloudy gray fluid. *Microscopic:* Examination showed the skin with coil glands, sebaceous glands and hair follicles. The subcutaneous tissue at one

are two types, classified as pericanalicular and intracanalicular. In the former, according to de Cholnoky, fibrous tissue surrounds the ducts and acini forming definite circumscribed tumor masses, increasing in quantity and choking the

epithelial cells. These cells undergo atrophy from pressure, deform, and finally disappear, so that nothing remains but fibrous areas arranged in concentric formation. Some of our cases showed exactly this pattern; and to illustrate that the two lesions may deceive even the elect, Case No. 2 in Table 1 is the same as Case No. 16. On the first admission only a small "lump" was removed from the lateral side of left breast; pathological diagnosis: gynecomastia. The lesion continued to grow, and nine months later mastectomy was done and the pathological diagnosis was fibro-adenoma. The intracanalicular fibro-adenoma are not so confusing as the fibrous tissues grow into the lumen of the duct pushing the epithelial lining before it. It would be interesting to know if any of de Cholnoky's thirty-six cases of fibromas or fibro-adenomas have recurred or if complete mastectomy was done at the time the original specimen was obtained. Charache[5] has stated that once gynecomastia develops it is persistent, and this assertion is partially substantiated by our one patient who was incompletely operated upon originally.

Trauma is mentioned very prominently by most authorities—de Cholnoky,[1] Charache,[5] Horsley,[6] and Christopher[7]—as the etiological factor responsible for the development of fibrous tumors and simple hypertrophy in the male breast. However, in our cases this has not been true. Of the fourteen patients with gynecomastias, twelve gave a history of "no trauma" to breast prior to onset or coincident with onset of symptoms; two gave a definite history of trauma. All suffered a dull, aching pain increased by the irritation of the military pack strap. The irritation of the pack strap caused the soldier to seek surgical relief. It may be suggested that the irritation was the trauma that produced the lesion; however, the tumors were all unilateral, nine in the right breast, five in the left breast, and the pack strap, when fitted properly, exerts equal pressure on each side of the anterior chest wall.

CASE REPORT

Pvt. D. Y. M., age nineteen, Hospital Register No. 15,035 (Case No. 7, Table 1), was admitted to Station Hospital August 4, 1941, with chief complaint of tumor mass in left breast. The soldier stated he had noticed a swelling in the left breast since the age of fifteen, his first year of high school. The "swelling" gradually enlarged to the present size. At times when he lifted heavy articles he suffered a dull pain in his left breast.

The general physical examination revealed nothing abnormal except enlargement of the left breast. On examination of the left breast a diffuse homogenous enlargement about the size of an orange was noted. No lumps or tender areas were present. The nipple was erect and there was no discharge therefrom. The axillary lymph-nodes were not palpable.

Laboratory reports, including urinalysis, blood serology, complete blood picture, and x-ray of chest were all within the limits of normal. On August 12, 1941, left mastectomy was performed through a crescent-shaped incision beneath the nipple.

Pathological report revealed the specimen to consist of a discoid mass of fibrofatty tissue 10 by 9 by 3 cm. The specimen was not covered by a membrane or capsule of any kind. It was composed of semifirm, pale gray tissue that resembled fibrous tissue in structure. There was nothing grossly recognizable as glandular tissue. The dense fibrous tissue was surrounded by a layer of fat. *Microscopic:* Examination showed a mass of very dense fibrous tissue containing relatively few neuclei. Scattered through the tissue were a few small lumps of glandular spaces, each lined by an irregular layer of columnar or cuboidal epithelial cells. The epithelial cells were adult in type and no mitotic figures were seen. Surrounding each gland space was a thin layer of relatively loose cellular fibrous tissue. A few ducts lined by epithelium similar to that already described were noted. Diagnosis: gynecomastia.

OPERATIVE TECHNIC

All of the cases have been handled in a similar manner. Mastectomy is done through a crescent-shaped incision beneath the nipple. The entire breast tissue is removed from the pectoralis fascia, taking precaution to conserve the nipple. If the

tumor is quite large, extensive dead space may be formed after its removal in which serum may collect. In this type of case a drain placed over the pectoralis muscle and brought out through a stab wound in the axilla has prevented the wounds from breaking down in any of our cases.

DISCUSSION

The etiology of gynecomastia and fibrous neoplasms of the male breast is obscure. Endocrine disturbances have been suggested as a basis for their development; however, in such an instance one would expect the lesion to be bilateral. Charache states that cases of gynecomastia have developed following prostactectomy. None of our cases had been previously subjected to this surgical procedure and none had any symptoms referable to the prostate. There was no complicating or co-existing disease in any of the cases of our series. Further, there was no evidence of glandular dysfunction; however, basal metabolism rates were not obtained in all of the cases.

Simple mastectomy is the treatment of choice for non-inflammatory tumors of the male breast no matter how innocent the lesion may appear, as it is well known that once gynecomastia develops the lesion remains permanently.[5] Further, carcinoma and sarcoma have both been observed to develop subsequent to gynecomastia and fibroma of the male breast.[5,8]

SUMMARY

1. Twenty benign lesions of the male breast have been reviewed.

2. One unusual case of lipoma of the male breast has been described in detail.

3. Trauma was found to be an etiological factor in only two out of fourteen cases of gynecomastia.

4. Average duration of symptoms prior to operation in the fourteen cases of gynecomastia was three and one-half years.

5. The lesion was found nine times in the right breast and five times in the left breast.

6. The etiology of gynecomastia is obscure.

REFERENCES

1. DE CHOLNOKY, TIBOR. Benign tumors of the male breast. *Am. J. Surg.*, 30: 298, 1935.
2. GUY, CHESTER L. Breast tumors in children. *Am. J. Surg.*, 33: 135, 1936.
3. WHITE, RICHARD JOSEPH. An unusual case of lipoma of the pectoral region simulating gynecomastia. *J. A. M. A.*, 88: 1146, 1927.
4. BOYD, WILLIAM. Surgical Pathology, 4th ed., p. 533. Philadelphia, 1938. W. B. Saunders Co.
5. CHARACHE, HERMAN. Tumors of the male breast. *Surgery*, 7: 889, 1940.
6. HORSLEY, JOHN S., JR. Benign and malignant lesions of the male breast. *Ann. Surg.*, 109: 912, 1939.
7. CHRISTOPHER. Textbook of Surgery, 2d ed., p. 889. Philadelphia, 1939. W. B. Saunders Co.
8. NEAL, PINSON M. Malignant tumors of the male breast. *Arch. Surg.*, 27: 427, 1933.

THE SURGICAL TREATMENT OF INTRACRANIAL
HEMORRHAGE COMPLICATING ECLAMPSIA

LIEUT. COMDR. WALTER D. ABBOTT, M.C., U.S.N.R.

DES MOINES, IOWA

THE occurrence of intracranial hemorrhage, either intracerebral or subdural, as a complication of eclampsia has not been recognized frequently in the past. In 1941, the author[1] reported a case of intracerebral clot complicating eclampsia in which surgical removal was rewarded with recovery. The basis of this report adds a subdural hematoma relieved by evacuation of the clot.

Eclampsia is a clinical entity which is not satisfactorily defined from either an etiologic or a pathologic point of view. Williams[2] states that eclampsia is an acute toxemia usually, but not always accompanied by convulsions. However, DeLee[3] remarks that this condition is not a disease but merely a symptom of some underlying cause, and he further mentions that cerebral hemorrhages of varying size frequently occur in eclampsia with this finding present in 40 per cent of autopsy material. Slemons,[4] in 1907, reviewed the literature to that date and Liebmann,[5] in 1925 quoted Schmorl's definition of eclampsia as "degenerative kidney changes particularly in the convoluted tubules where albumin and fatty alterations are observed: necrosis in the liver with thrombosis in the interlobular and intralobular vessels; degeneration in the heart, particularly in the muscles; hemorrhage and softening into the brain; and a widespread thrombosis in the internal organs."

Bell[6] more recently described a characteristic glomerular lesion of the kidney consisting of dilation of the glomerulus and thickening of the capillary basement membrane with narrowing of the lumen of the capillaries. Of the forty-six cases that have been reported, Slemons found the reports of seven and added two more in 1907. The next review was that of Schmidt,[7] in 1911,

who brought the total to twenty-four. Caffier[8] found thirty-eight cases up to 1927 with four others that had been discarded because of incompleteness either of the history or of the autopsy. His paper brings the literature to that date. Since then, similar conditions have been reported by Liebmann, Pohl,[9] Bock[10] and Wronski.[11] Bock adds also the case of Berg and mentions three others as well as the case of Thalheimer which cannot be included because of lack of necropsy evidence. Knight,[12] in 1925, added a case in which there was hemorrhage into the frontal lobe, and King,[13] in 1933, reviewed the literature bringing the total to forty-six cases. However, only thirty are carefully analyzed in his report because of inadequate records. The age limit was from eighteen to forty-three years and the parity from primigravidity to tertiodeciparity, with a distribution of nineteen primiparas and eleven multiparas. Antepartum cases numbered fourteen, the earliest of which was at three months, and ten occurred during delivery and six during the puerperium.

These proportions are similar to those reported by Greenhill for the distribution of true eclampsia. Of these cases only fourteen showed true premonitory signs. This amazingly low figure makes the seriousness of the situation all the more apparent. It is not impossible that symptoms would have been observed in others had they been looked for, but this fact emphasizes again the necessity for the strictest watchfulness by those attending patients in confinement.

Headache was the most common symptom followed by vomiting and edema. Visual disturbances occurred in only seven instances and epigastric pain in six instances. Albumin in the urine was reported

fifteen times, and an elevation of temperature was observed in fifteen cases. In nine instances circulatory collapse was the first warning of anything abnormal, and in eight cases unconsciousness with or without cardiac signs ushered in the condition. The blood pressure records have not been carefully taken in all instances, particularly in the reports from Europe. The description of cerebral bleeding into the brain is the significant part of the pathologic condition which has been confirmed by these various reports. However, the type of hemorrhage will vary from a petechial to a massive hemorrhage into the frontal or occipital lobe on into the internal capsule, and in one case into the pons. One not infrequently sees this hemorrhage occur in the absence of convulsions, and Benda[15] believes that the increased capillary permeability is due to toxemia.

The two cases reported by Cronin and Knight are the only ones apparently in which recovery from massive hemorrhage occurred, and in neither instance did the patient have convulsions. King states that in nine cases exclusive of his report massive hemorrhage into one or the other parts of the brain is reported. These are the reports of various authors cited in his extensive review of the literature. The fact that such a condition occurs sounds a warning note to prevent or at least attempt to combat such a condition when it does occur.

The following report is the only one in which surgical removal of the clot has been rewarded by recovery of the patient:

CASE REPORTS

CASE I. Mrs. R. H., aged nineteen, had been seen at the antepartum clinic since October, 1940, by Dr. Eugene Penn. This was her first pregnancy and each time she visited the clinic her condition was satisfactory until March 10, 1941, at which time it was noted that she had gained 9½ pounds (4,300 Gm.) in the previous week, that her blood pressure had risen from 120 systolic and 80 diastolic to 130 systolic and 100 diastolic and that the urine contained a considerable amount of albumin. The patient stated that for four days she had suffered from a severe headache accompanied by spots before her eyes and that there was some swelling of her feet and legs. On March 13, she entered the hospital complaining of headache in the occipital region and was in labor that lasted for ten hours. Light ether anesthesia was administered and delivery of a normal boy weighing 8 pounds (3,630 Gm.) was effected by low forceps delivery. She was given 2 cc. of 50 per cent magnesium sulfate intramuscularly and 500 cc. of 20 per cent dextrose intravenously. After the anesthesia the patient remained stuporous, pale, cold and clammy. The pulse rate ranged from 120 to 140 a minute and the respiratory rate from 28 to 30 a minute; the blood pressure remained at 140 systolic and 102 diastolic.

At no time did she show any evidence of a convulsion. However, her pupils were dilated and did not react to light, and there was a homonymous hemi-anopia to the left with a slow horizontal nystagmus. Ophthalmologic examination revealed a 5 diopter choked disc on the right, 3 diopters on the left with a homonymous hemi-anopia.

Roentgenograms of the skull were negative and neurologic examination was negative except for the profound stupor. The temperature remained at 101°F., and the blood pressure at 140 systolic and 102 diastolic. The pulse rate was 60 a minute and the respiratory rate was 14 a minute. Examination of the urine revealed an acid reaction, the specific gravity was 1.024, albumin was 6.5 Gm., there were occasional pus cells and the blood urea nitrogen was 28 mg. per hundred cu. cm. Examination of the blood otherwise was negative, as was the Wassermann reaction.

Hypertonic dextrose was administered daily by venoclysis. She was referred to the neurological section of Iowa Methodist Hospital where a diagnosis of a lesion of the right occipital lobe was made and I believed that it was questionable whether this was the result of an intracerebral hemorrhage or a preexisting glioma with hemorrhage into the tumor. Two days later, under anesthesia of avertin with amylene hydrate, ventricular studies were attempted. Perforator openings were made over the occipital regions of the skull and when a trocar was passed into the right occipital lobe resistance was met at a depth of 3 cm. Aspirations from the trocar resulted in the removal of clots of old blood. A trocar was then passed into

the left lateral ventricle and clear fluid escaped under pressure. A total of 60 cc. of fluid was replaced with an equal amount of air, and roentgenograms revealed a shifting of the ventricular system to the left and a failure of filling of the posterior horn of the right lateral ventricle. An osteoplastic flap was then turned over the right occipital area and the brain was found to be under a significant increase of pressure with a flattening of the convolutions of the occipital lobe. The cortex was incised and a blood clot weighing 8 Gm. was removed.

After removal of this clot there was bleeding from the small branch of the posterior cerebral artery, which was controlled with electric coagulation. A sample of tissue taken from the surrounding area was examined by Dr. R. F. Birge, who stated that there was no evidence of tumor. The patient made a satisfactory recovery and on January 26, 1942, stated that she had no symptoms except an inability to see to the left. Her vision was 20/20 in the right eye and 20/25 in the left eye, and the choked discs had completely receded. However, there was a residual homonymous hemi-anopia on the left side. The blood pressure was 120 systolic and 80 diastolic and urinalysis was negative with blood urea nitrogen 14 mg.

A careful review of the literature revealed no evidence of subdural hematoma being recognized clinically as a complication of eclampsia, and in a review of 105 subdural hematomas in my experience there was no evidence of such a condition until the present case.

CASE II. Mrs. A. T., a negress, age twenty, was transferred to the neurosurgical service of Mercy Hospital by Dr. I. H. Odell on November 11. Her second baby was born July 8th in Broadlawns General Hospital. The prenatal course was negative until she began to develop a moderate edema during the last two months of pregnancy. The blood pressure at the beginning of the sixth month was 98 systolic and 60 diastolic; it gradually rose and at the ninth month it was 128 systolic and 98 diastolic. There was a trace of albumin in the urine on the second of July and two plus albumin on July 8th, the day of delivery. On this day, the patient complained of severe generalized headaches and blind spells. The duration of the delivery was 3½ hours. Forty-five minutes after the delivery the patient had a clonic type

of convulsion beginning in the hands and involving the entire body, lasting four minutes. The blood pressure on the date of delivery was 165 systolic and 100 diastolic; on the second day it was 180 systolic and 100 diastolic, and it gradually subsided until on the tenth day the blood pressure was 132 systolic and 80 diastolic. The urine was clear and her postnatal course was negative, and she was discharged on the eleventh postpartum day.

Approximately a month before her admission to Mercy Hospital on November 11, 1941, she developed severe headaches which required the administration of morphine and spinal puncture revealed increased pressure. The fluid was not examined at that time. She obtained relief until approximately a week before her admission to the hospital on November 11, 1941, when her headaches recurred with intense severity. Electro-encephalographic tracing revealed increased large delta waves over the right frontal and left occipital lobes. Neurological examination was negative. Spinal puncture revealed clear fluid under pressure of 30 cm. of water; cell count 7; total protein 30 mg.; colloidal gold 1110000000, Wassermann test of the fluid was negative. Pneumo-encephalography revealed a filling defect over both cerebral hemispheres in the frontal area, greater on the right than on the left. Under avertin anesthesia with amylene hydrate, an incision was made in the right superior temporal region and a perforator opening was inserted into the bone. The dura was discolored and under tension. When this was incised there was an escape of yellow fluid and old blood and following this the brain began to pulsate. There were similar findings on the opposite side.

The patient made a satisfactory recovery and when seen on January 19, 1942, there were no complaints. A careful inquiry revealed no history of previous injury.

CONCLUSION

Intracranial hemorrhage undoubtedly is a more frequent complication of eclampsia than has hitherto been realized and prompt recognition, with proper surgical intervention when indicated, may lead to complete recovery. These two cases are reported in the hopes that such a condition will always be borne in mind and that the patient with eclampsia will receive the benefit of

adequate ophthalmologic and neurologic consultation.

REFERENCES

1. ABBOTT, W. D. The surgical treatment of intracranial hemorrhage complicating eclampsia. *J. A. M. A.*, 117: 1439–1440, 1941.
2. WILLIAMS, J. W. Obstetrics. 6th ed. New York, 1931. D. Appleton & Co.
3. DELEE, J. B. Principles and Practive of Obstetrics. Philadelphia, 1938. W. B. Saunders & Co.
4. SLEMONS, J. M. Eclampsia without convulsions. *Bull. Johns Hopkins Hosp.*, 18: 338, 1907.
5. LIEBMANN, S. Eklampsie ohne Krämpfe. *Zentralbl. f. Gynäk.*, 49: 1906, 1925.
6. BELL, E. T. Renal lesions in the toxemias of pregnancy. *Am. J. Path.*, January, 1931.
7. SCHMIDT, H. H. Eklampsie ohne Krämpfe und ohne Bewistlosigkeit. *Ztschr. f. Geburtsch. u. Gynäk.*, 69: 143, 1911.
8. CAFFIER, P. Retrspecktive Diagnose auf Eklampsie ohne Krämpfe. *Med. Klin.*, 23: 162, 1927.
9. POHL, R. Ploetzlicher Tod in der Schwangerschaft. *Zentralbl. f. Gynäk.*, 51: 1913, 1927.
10. BOCK, A. Klinischer Beitrage zyr toedlichen Gestose ohne Krämpfe. *Zentralbl. f. Gynäk.*, 52: 102, 1928.
11. WRONSKI, M. Zur Eklampsie ohne Krämpfe. *Zentralbl. f. Gynäk.*, 52: 1528, 1929.
12. KNIGHT, L. C. Eclampsia with cerebral hemorrhage in the puerperium. *Illinois M. J.*, 47: 53–54, 1925.
13. KING, A. C. Eclampsia without convulsions terminating in cerebral apoplexy. *J. A. M. A.*, 100: 15–16, 1933.
14. SCHMORL. *Arch. f. Gynäk.*, 50: 727, 1926.
15. BARRETT, C. W. and HEAGER, S. B. A consideration of eclampsia with a case accompanied by hemiplegia. *J. Obst. & Gynec.*, 64: 463, 1905.

꘠

SALVATION of the malignant uterus for purpose of future pregnancy is condemned in every particular.

From "Vaginal Hysterectomy" by James William Kennedy and Archibald Donald Campbell (F. A. Davis Company).

ACUTE PHLEGMENOUS GASTRITIS WITH MULTIPLE PERFORATIONS*

CASE REPORT

J. ROSENTHAL, M.D.   AND   M. J. TOBIAS, M.D.
Pathologist, Welfare Hospital for Chronic Diseases   Assistant Attending Surgeon, Third Surgical Division, Welfare Hospital

NEW YORK, NEW YORK

ACUTE perforations as a first manifestation of gastric ulcer attain in some instances an incidence of 50 per cent. Acute simultaneous multiple gastric perforations, however, are much rarer, and as a first manifestation attain an incidence of only 11.1 per cent; for this reason alone they deserve a recording.

CASE REPORT

F. S., an eighteen-year old, white male of German parentage, was admitted from a New York City Hospital to Welfare Hospital on March 19, 1940. The patient was the second of two children. A brother, two years older, is said to be of normal development and in good health. As an infant, the patient was slow in development. Thus, although he spoke at one and one-half and walked at two years of age, his walking was cautious and slow. He tired easily and "fell over quickly" when brushed against or when caught off balance. The patient was a poor eater and always had sluggish bowels. He was subject to frequent colds. The only children's diseases recorded were measles and chickenpox. The family history was entirely irrelevant. At nine and one-half, the patient commenced to make the rounds of various city institutions because of a slowly developing progressive weakness of the lower extremities. For some six years prior to his last admission, the patient was unable to walk and had been hospitalized in various institutions, and finally transferred to Welfare Hospital.

It is of interest to note that the physical findings of this patient as recorded below do not differ in any essential respect from those recorded at a New York City hospital, three years prior. It might also be well to mention

that during his stay at the latter hospital, the patient had some six attacks of "loose bowels, accompanied by anorexia, and in several instances by tachycardia, but without mucus or blood. These were easily controlled by simple astringents."

Physical examination upon admission revealed a red-haired, grey-eyed, wasted and markedly deformed white male, co-operative and apparently comfortable. The skull was somewhat deformed and teeth carious and indented. The lower jaw protruded so that the teeth could not be approximated. The spine was immobile, nontender, and showed a marked thoracolumbar scoliosis to the left with lordosis. The thighs were outwardly rotated and acutely flexed at knees and there were also equinovarus deformities of feet, so that the patient assumed an "Indian squatting" position. The elbows and knees could not be positively extended to a 90 degree position. There was marked atrophy of the trapezii, pectorals, and of the muscles of the shoulder girdle, back, arms, forearms, thighs and buttocks; while the muscles of the calfs and temporal regions appeared hypertrophied. The skin of trunk, arms, and face showed numerous melanotic papules as well as a mild acne vulgaris. The tongue was large (macroglossia) and its papillae hypertrophied. The pharyngeal tonsils were hypertrophied. The thyroid was not enlarged; the salivary glands appeared normal, and there was no adenopathy noted. The radials, carotids and femorals were small. The lungs and heart were essentially negative. The blood pressure was 110/78 and the pulse rate 90. There was little subcutaneous fat, and the spine and sacrum were palpable through the left abdominal quadrants. Liver, spleen, and kidneys were not palpable. The external genitalia appeared of normal development; anus and rectum showed

no pathological condition. The deep reflexes were absent. The superficial reflexes were present but more active on the left side. Ab-

FIG. 1. Stomach Large hemorrhagic area in cardiac end of stomach reaching almost to esophagus.

normal reflexes were not elicited. There were no signs of meningeal irritation. The sensory reflexes for pain, touch, temperature, etc., were normal. The patient retained some degree of motion of the fingers, shoulders and toes. He was able to turn his head weakly and at times barely to raise the extended head. He could not rise from the horizontal plane, and when placed in a sitting position he rested upon the left buttocks. The cranial nerves were uninvolved with the sole exceptions of the seventh (weakness of facial muscles) and the eleventh (weakness of trapezii and sternomastoids). Co-operation was good. There were no illusions, hallucinations or delusions. The patient was oriented as to time, place and person; he was able to do simple calculations and his memory was good

X-ray examination showed the bones of the skull (inclusive of sella turcica) to be normal. The bones of the wrists and hands showed a

marked degree of decalcification. The mandible revealed infected root fragments in the right molar region but otherwise no abnormalities. Atrophy and decalcification were also present in the bones of the shoulder girdle, of the legs, and of the lower ends of the femora. The lungs showed no pathological condition upon x-ray examination. The urine was negative. The blood showed: hemoglobin 96 per cent; red blood cells 3,896,000; white blood cells 8,650; sugar 69 mg. per cent; creatinin 2.4 mg. per cent. The blood Wassermann was negative. The diagnosis was pseudohypertrophic muscular dystrophy.

Apart from his disabilities, the course of this patient's illness was uneventful until July 4, 1940. Between that date and August 3, 1940, the patient had several episodes of "loose bowels." These apparently were of the same nature as that experienced by the patient on previous occasions. They were unaccompanied by tenesmus, abdominal tenderness, fever, mucus, or blood, and they were apparently easily controlled by astringents.

On August 3, 1940, however, the patient had an accession of abdominal pain centered about the umbilical region. On examination, he appeared pale and distressed; he was nauseated but did not vomit. Pulse was 72 and temperature normal. The patient localized the pain just above the umbilicus, and described it as a dull ache without radiation. The abdomen was soft; spasticity, tenderness, and distention were absent. The colon was readily outlined through the soft abdominal wall and contained large amounts of movable material, apparently feces. Rectal examination showed a flaccid sphincter, and the entire ampulla filled with moderately firm feces. Oil enemas and manual disimpaction followed later on by a few voluntary bowel movements resulted in apparent improvement, so that on August 5 and 6, the patient was free of pains, had no nausea, was hungry, and the abdomen remained soft and flat.

However, on the evening of August 6, the epigastric pains returned and were accompanied since the morning of August 7 by persistent vomiting of coffee ground material. When seen in surgical consultation with Dr. J. H. Morris on the same morning, the patient appeared acutely ill. The abdomen was moderately distended. There was slight generalized and marked epigastric tenderness. There

was a curious absence of muscle spasm and rigidity. The liver and spleen were not palpable. The liver dullness was obliterated and there

blood and stomach contents were seen to ooze. One area of necrosis measuring about 6 cm. in diameter was situated at the lesser curvature,

FIG. 2. Large masses of round cells extending through entire width of mucosa Marked fibrosis at A.

was a shifting dullness in the flanks. Rectal examination at this time showed tenderness high up in the midline, and an abdominal x-ray plate revealed a pneumoperitoneum and marked elevation of the right diaphragm "secondary to perforation in the gastrointestinal tract." The vomitus showed a four plus Benzidine, some red blood cells, and a trace of bile. The white blood count at this time was 20,450 (polymorphonuclears 69 per cent), the urine showed acetone and diacetic acid, temperature 100°F. and pulse 130. The preoperative diagnosis was perforated gastric ulcer and spreading peritonitis. Immediate laparotomy was agreed upon.

Cyclopropane anesthesia was used. On opening the peritoneum, the hiss of escaping gas was heard. There was a considerable quantity of free, thin, reddish-brown fluid.* All peritoneal surfaces showed signs of acute diffuse inflammation, and in many areas the peritoneum was lifted from its bed by small emphysematous bullae. The loops of the small intestine were collapsed. The stomach was enormously distended and contained a huge quantity of soft crepitant material as determined by palpation. Inspection of the stomach revealed two large areas of frank necrosis through which free

* Cultures of this fluid remained free of colonies after fourty-eight hours' incubation.

well above the pyloric region. The second area of necrosis was located on the greater curvature of the stomach at the junction of its lower and
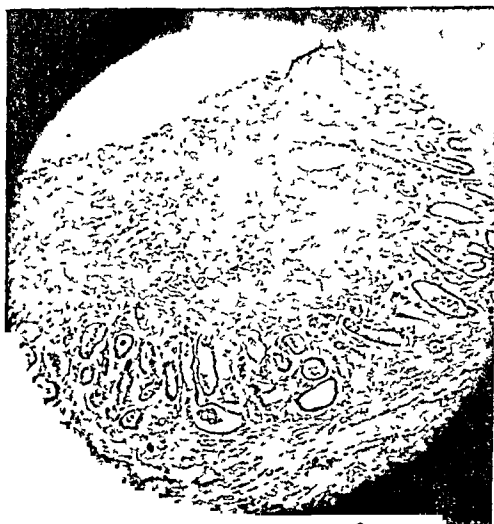

FIG. 3. Circumscribed degeneration of part of the mucosa.

middle thirds. Its long axis was in the direction of this curvature. Part of the great omentum was plastered against this perforation and covered the same imperfectly. This was dissected away without difficulty and appeared

to consist of recent adhesions. Both areas were dark grey in color and mushy to the touch. Gas exuded from them and dissected its way

hours of what appeared to be continuous improvement (the temperature had fallen to 99°F. and the pulse rate to 108), the patient
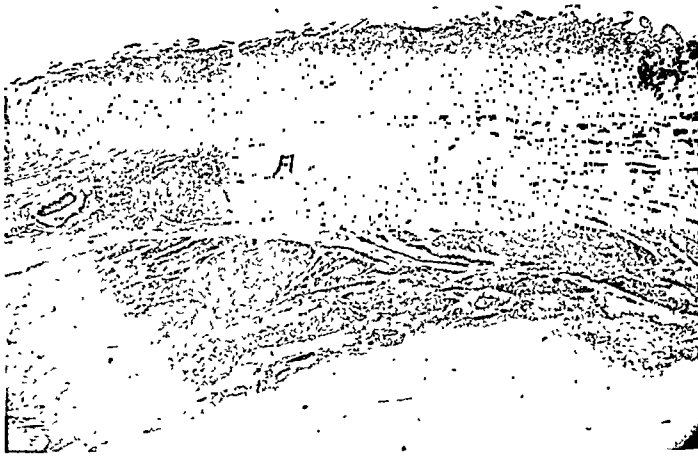

FIG. 4. Suppurative process involving principally the submucosa (A).

upward into the surrounding serosa and under the adjacent omentum.

In addition to these large areas of necrosis, there were four to five smaller areas of the gastric wall varying in color from red to dark purple which were not as severely involved. The liver and gallbladder appeared normal. Despite a 500 cc. blood transfusion which was being administered during the operation, the patient was doing very poorly. In view of this, no major procedures could be undertaken, and the operation of choice appeared to be a simple infolding of the necrosed areas. Accordingly, the peritoneal cavity was aspirated dry and the involved areas were invaginated by a double row of Lembert sutures. Because of the absence of induration along the involved areas, this could be accomplished without much tension. The great omentum was then tacked over the suture lines as accurately as possible. Two cigarette drains were placed, one in the lesser sac, and another in the left lumbar gutter, and the operation terminated, after the customary layer-by-layer closure of the abdominal wall.

The patient was returned to the ward in partial shock. For the following three days, the temperature varied between 101 and 101.8°F., pulse 132 to 140, and respirations 24 to 28. On August 10, 1940, the third postoperative day, it was noted that the abdomen was soft and not distended, the suture line intact, and that the abdominal wound appeared normal. On August 11, 1940, after twenty-four

developed difficulty in breathing, and signs of consolidation in the lower left lobe. The blood count at this time showed: red blood cells 5,350,000; white blood cells 8,350; urine p.H 7.5.1010, with traces of sugar and acetone, and hyaline and granular casts. Respirations became labored, cyanosis ensued, and he expired at 11:30 P.M. of the same day, apparently of respiratory failure.

For the sake of brevity, the macro- and microscopical findings only of the stomach will be given in detail. However, for the purpose of completeness, it should be stated that the remainder of the gastrointestinal tract showed no pathological condition. The brain weighed 1,460 Gm. The surface showed no marked abnormalities. Sections were taken through the medulla, pons, midbrain, cerebellum and cortex and stained by hematoxylin and eosin, Nissl and myelin stains. None of these sections showed any definite inflammatory or degenerative changes. As a matter of fact, the only other pathologic lesions found were: atrophy and degeneration of the voluntary muscles, generalized peritonitis, toxic hepatitis, atelectasis of the left lung and bilateral pleural effusion.

The esophagus was opened and showed no gross lesions. Along the greater curvature on the anterior surface of the antrum were suture lines. When these were opened, the greater part of the antero-lateral part of the fundus was found to be necrotic. (Fig. 1.)

A large portion of the fundal mucosa was the site of a diffuse hemorrhagic infiltration. There were also numerous smaller areas 1 to 3 cm. in diameter showing small hemorrhagic infiltration which were sharply set off from the mucosa. Some of these showed early necrosis with softening and ulcer formation. The pyloric end of the stomach and duodenum showed no gross abnormalities.

The stomach showed changes varying from early inflammation to complete gangrene of the entire width of the stomach wall.

Proceeding from the pyloric end of the stomach to the fundus, where the involvement was greatest, the following changes were noted in the layers of the stomach wall:

At the periphery of the involved area, the mucosa appeared generally normal. In areas, however, large masses of lymphocytic tissue were found. In one such area, a large collection of round cells was found and was seen to extend from the submucosa through the entire width of mucosa to its internal surface. This focus which was composed mostly of lymphocytes, contained also mononuclears and plasma cells. Thick strands of connective tissue were seen within this cell mass. (Fig. 2.) As one proceeded to the center of the lesion, one noted first congested vessels, later cellular increase and tissue degeneration. In one such area, the region of degeneration assumed a circumscribed appearance, involved only the upper layers of the mucosa and took an eosinophilic stain. This sharply outlined region suggested necrosis associated possibly with peptic digestion. (Fig. 3.) No limiting zone of inflammatory cells was found, the process apparently having been recent and acute. In the regions of gross necrosis, the mucosa was found to be in a state of marked degeneration so that only the shadowy remains of original tissue were outlined. In such regions there was seen complete exfoliation of the mucosa apparently due to recent necrosis.

The involvement of the submucosa was an outstanding feature of the pathological state in this organ; in fact, the purulent process appeared to spread in the loose tissues of this layer. (Fig. 4.) In the less affected regions, the only abnormalities seen were increase of connective tissue and hypertrophy of the walls of the small arteries. Further one encountered marked congestion, hemorrhage and focal collections of inflammatory cells. The amount of inflammatory exudate, at first noted as a beginning abscess, was seen to spread in all directions but involved especially the sub-

Fig. 5. Purulent process in submucosa and muscularis; perforation through stomach wall, exudate on serosa.

mucosa itself. Upon one section there was found a round collection of inflammatory material surrounded by a thin fibromuscular wall, the entire appearance suggesting suppuration in a vessel, probably a vein. The advance of the purulent process finally resulted in an extension through the entire stomach wall with formation of a purulent exudate in the serosa. In the central area of gangrene, all cellular architecture had disappeared.

The muscularis was involved by the adjacent suppurative process but to a somewhat lesser degree. In the areas of perforation of course, the muscle fibers were completely degenerated. There was no evidence of any marked antecedent pathological condition in the muscle fibers.

The degree of involvement of the serosa varied with its proximity to the central gangrenous area. At the periphery of the lesion only a comparatively few cells, particularly lymphocytes, were seen. Near the central area of involvement a frank purulent exudate was found. (Fig. 5.)

COMMENT

The violent necrobiotic process in this case bears a resemblance to infarction. However, a careful search of all the vessels in the vicinity failed to disclose thrombosis or occlusion of any vessel.

Of interest in this case is the finding of numerous cell aggregates consisting principally of lymphocytes. In some areas these resemble the lymphoid follicles seen in the gastrointestinal tract generally, except that they do not have the regularity of outline nor do they have a lighter staining germinal center. Some of these collections are particularly large, their cells spread in all directions, and they contain areas of fibrosis. Some others of these areas resemble those of lymphocitic hyperplasia seen in chronic infections. This in itself is interesting.

M. Letulle[3] reported the presence of lymphoid nodules in the walls of the stomach. He was of the opinion that this finding might offer an explanation for the occurrence of such conditions as perigastric tuberculous abscess, perforation of gastric cancers, interstitial abscess and *circumscribed phlegmonous gastritis.*

B. Cuneo[4] in a masterly survey of the entire subject of the lymphatic system of the stomach, describes and portrays two kinds of lymphoid aggregations: encapsulated lymphoid follicles placed immediately above the muscularis mucosae, and diffuse lymphoid elements which surround the secretory glands. In this case, the presence of large masses of round cells *accompanied by fibrosis* would seem to indicate some form of chronic irritation. The pathogenesis of the lesion, however, is not entirely clear. A possible explanation that suggests itself, particularly from the violence of the process[*] and the absence of any vascular obstruction, is acute peptic digestion. Whether this process was initiated by some form of chronic inflammation in the mucosa or by another factor such as avitaminosis, is unknown; however, once begun, it extended through the loose tissues of the submucosa. The end result was a hemorrhagic and gangrenous process of the gastric wall with massive perforations and peritonitis.

SUMMARY

A case of acute phlegmenous gastritis in a patient with muscular dystrophy is presented.

The pathological processes as found at operation and at autopsy are described and some of the microscopical sections are submitted; from these, however, no decisive clue as to the pathogenesis of the lesions could be adduced.

The evidence on hand indicates[*] that this case does not belong to the class of similar cases of massive perforation and/or gastromalacia of the stomach and other parts of the upper gastrointestinal tract reported as occurring in association with various lesions of the brain.[5,6] Less certain, however, is the possibility that it may belong to the group of cases of "neurogenic gastric ulcer without primary intracerebral disease" as recently reported by Boles and Riggs.[7]

Finally, the absence of muscle spasm and rigidity in the presence of an acute peritoneal inflammation is pointed out as a curious concomitant of this case.

REFERENCES

1. TROUTT, JAMES H. Acute perforation of gastric ulcer. *Surg., Gynec. & Obst.,* 47: 815–821, 1928.
2. MASSON et al. Simultaneous perforation of multiple ulcers. *Minnesota Med.,* pp. 289–294, May, 1927.
3. LETULLE, M. Ganglions lymphatique parietaux de l'estomac. *Bull. et mém. Soc. d. anat. de Paris,* 74: 1093–1095, 1899.
4. CUNEO, B. De l'envahissement du system lymphatique dans le cancer de l'estomac, etc. Theses de Paris, 1900.
5. CUSHING, HARVEY. Peptic ulcer and the interbrain. Fourth Balfour Lecture, April 8, 1931, University of Toronto.
6. OPPER, L. and ZIMMERMAN, H. M. Ulcers of the digestive tract in association with cerebral lesions. *Yale J. Biol. & Med.,* 11: 49–84, 1938.
7. BOLES and RIGGS. Gastric ulcer. *J. A. M. A.,* 115: 1771, 1940.

---

[*] Indeed, the destruction of parts of the gastric wall was so complete that corrosive poisoning was suggested. However, negative findings upon chemical analyses of portions of the stomach and gastric contents excluded this possibility.

[*] Sections of the brain submitted to study failed to yield any evidence of a pathological disorder. We are indebted to Dr. Stevenson of New York University, College of Medicine, for a review of these slides.

PREVESICAL HERNIA

H. Lewis Berson, m.d.

BROOKLYN, NEW YORK

A SUPRAVESICAL hernia located in the prevesical space of Retzius is a rare condition. Walker, in 1933, presented two cases and found only three others in the literature. His were the only reports in the American literature until Stalker and Gray, in 1939, added another, the only one encountered between 1910 to 1939 at the Mayo Clinic. Wervi and Orr, in 1940, added a fourth and contributed the information, after an exhaustive search of the literature, that in all there were thirty-eight authentic case reports and twenty-three doubtful ones. Their tabulation revealed twenty-three from the German clinics beginning with Linhart's report of one in 1856. That this condition was known prior to that time is proved by a plate appearing in Sir Astley Cooper's book published in 1804 in which six hernial openings, of which two are in the supravesical space, are demonstrated.

ANATOMY

The supravesical area is a triangular fossa limited laterally and above by the lateral umbilical ligaments (the obliterated hypogastric arteries) and below by the peritoneum as it passes from the anterior abdominal wall to the fundus of the bladder. This fossa is divided into two parts by the median umbilical ligament. Beneath the peritoneal reflection and lying between the pubic bone and the anterior wall of the bladder is the prevesical space.

NOMENCLATURE

The multiplicity of names applied to hernias in this region has added nothing but chaos and confusion to the subject. Wervi and Orr list twenty-two different titles applied to these hernias in the supravesical region. The simplest classification since they all arise in the supravesical area is to call them supravesical hernia and to add to this title other terms according to the direction the protrusion takes. These hernias generally take one of three courses: anteriorly, to appear on the exterior in the hypogastrium. These hernias offer no particular diagnostic problem. They may pass through an orifice in the superolateral zone between the lateral border of the rectus muscle and the lower lateral umbilical ligament to appear in the femoral region. Finally, the herniation may extend inferiorly into the prevesical space between the bladder and the pubic bone. This type is most rare and since it is hidden behind the anterior bony pelvis and does not come to the surface is most difficult to diagnose. An example of this form of supravesical hernia was recently encountered and because of its rarity deserves recording:

CASE REPORT

F. M., a male carpenter, forty-two years of age, was admitted to the Cumberland Hospital[*] on January 14, 1941. His chief complaint began eleven days previously with sudden, sharp epigastric pain that radiated downward to the suprapubic region and to the right lower quadrant. The pain continued the following day and was accompanied by a watery diarrhea. This attack wore off and he was symptomless for four days. At this time he had another severe attack of pain that doubled him up but only lasted for a few hours. He was again free of any symptoms until 9 P.M. of the night of admission at which time his pain came on suddenly in the periumbilical region. Nausea but no vomiting was present. He stated that when he attempted to straighten up the pain was in the umbilical area but when he bent over the pain seemed to be in the suprapubic region. There were no urinary symptoms.

He appeared acutely ill but his temperature, pulse and respirations were normal. He kept

* Surgical service of Dr. John J. Gainey.

his legs flexed believing that he had less pain when in that position. No distention was present nor were any masses felt. Marked tender-
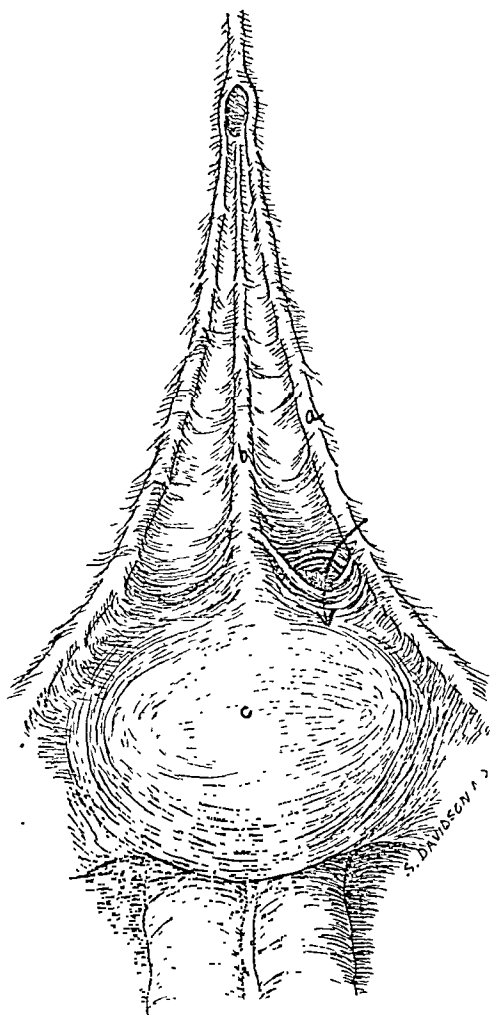

FIG. 1. Supravesical hernia leading to the prevesical space: (a) lateral umbilical ligament; (b) median umbilical ligament; (c) bladder.

ness was present in the lower quadrant particularly over McBurney's point. Right lower rectus rigidity and spasm were easily elicited. Rectal examination revealed only a firm, hard prostate gland. Catheterization caused severe pain. His urinalysis was normal. The white cells totalled 16,800 with 86 per cent polymorphonuclear cells. The preoperative diagnosis was acute appendicitis.

Operation was performed four hours after admission under a general anesthesia through a lower right rectus muscle retracting incision. The appendix was found to be retrocecal and

about 6 cm. in length. This did not seem to be sufficiently involved to present so severe a clinical picture. The incision was enlarged downward and the pelvis explored. An opening was noted in the anterior peritoneum just above the bladder and between the median and lateral umbilical ligaments. About 10 cm. of small intestine were incarcerated by the rim of this opening and lay in a sac measuring 6.5 cm. in length and extending down in the prevesical space. The incarcerated bowel was delivered and found to be dark red and hemorrhagic but viable. No resection was necessary. The sac was not removed but the opening was closed by a continuous chromic catgut suture. The patient made an uneventful recovery and was discharged from the hospital in eleven days.

COMMENT

The diagnosis is a difficult one to make. Some authors do not believe that the correct diagnosis can be made preoperatively. Wervi and Orr credit Fromme with making such a diagnosis before operation and he used a cystoscope to demonstrate a bulge in the anterior wall of the bladder.

The usual experience has been that these hernias were found accidentally in the course of a laparotomy for some other condition, as in the case here reported, or at necropsy. The clinical picture is ordinarily that of small intestinal obstruction with nausea, vomiting, distention and colicky pains. When these symptoms are accompanied by urinary frequency the possibility of such an internal hernia should be entertained. One may, however, be fooled in the case of elderly male patients with prostatic trouble. The use of the cystoscope to visualize a bulge in the anterior bladder wall is corroborative but may be a dangerous procedure in a sick patient. A scout film is of diagnostic aid and not as taxing to the patient.

The treatment is that for acute intestinal obstruction with repair of the hernial opening. Resection of bowel when necessary will increase the mortality and morbidity. Although some surgeons advocated mobilization and excision of the sac, obliteration of the opening by interrupted or continuous

suture is sufficient for a cure. No recurrences due to failure to remove the sac have been reported.

SUMMARY

1. Hernia into the prevesical space is a rare form of hernia.

2. Diagnosis before operation has rarely been made. But signs of intestinal obstruction and urinary frequency should be suggestive.

3. Cystoscopic examination is corroborative but must be used with care.

4. A scout film is of diagnostic help.

5. Resection of intestine if necessary will increase the morbidity and mortality.

6. Simple closure of the opening will provide a cure. Removal of the sac merely increases the risk.

REFERENCES

FINSTERER, H. Hernia supravesicalis interna incarcerata. *Med. Klin.*, 33: 295–297, 1937.

KOCHAJ, J. and WOLF, J. Supravesical preperitoneal hernia; 2 cases. *Polska gaz. lek.*, 13: 356–357, 1939.

KRAMERENKO, Y. Y. Hernia supravesicalis interna. *Novy khir. arkhiv.*, 30: 531–547, 1934.

OVNATANYAN, K. T. Hernia supravesicalis interna incarcerata. *Novy khir. arkhiv.*, 34: 409–412, 1935.

PICK, J. Anatomy of paravesical and supravesical hernias. *Arch. f. klin. Chir.*, 190: 553–568, 1937.

ST. JOHN, V. Properitoneal hernia. *West. J. Surg.*, 41: 183–190, 1933.

STALKER, L. K. and GRAY, H. K. Prevesical hernia. *Proc. Staff Meet., Mayo Clin.*, 14: 321–324, 1939.

SZABO, D. Supravesical hernia. *Orvosi hetil.*, 82: 224–225, 1938.

WAKELY, C. P. G. Prevesical hernia; case. *Med. Press*, 203: 37–38, 1940.

WALKER, I. S. Hernia into the prevesical space. *Ann. Surg.*, 97: 706–712, 1933.

WERVI, W. N. and ORR, T. G. Supravesical hernias. *Surgery*, 8: 312–325, 1940.

INTRAMUSCULAR INFILTRATION WITH METYCAINE FOR THE RELIEF OF SEVERE AND PERSISTENT POSTOPERATIVE BACKACHE

CASE REPORT

CAPTAIN EDWARD S. BREWSTER, M.C.

CAMP COOKE, CALIFORNIA

BACKACHE has been a troublesome experience to most individuals who have been confined to bed for any prolonged period of time. In many instances, the backache may have been so slight as to merely create a mild and transient type of annoyance, but in some cases the pain has been so distressing that opiates were required for relief.

Usually regarded as a minor and rather unimportant postoperative complication, backache has received scant attention in surgical textbooks and publications. For example, Warbasse and Smyth[1] state that, "Pain in the sacrolumbar region commonly complained of after operations can be relieved by placing a pillow in the hollow of the back." Cutting,[2] in his monograph devoted to preoperative and postoperative treatment, advocates as follows: "A small pillow placed under the head, under the normal lumbar curve and under the knees of a patient at the time of operation will do much to prevent the development of the condition. Nurses and assistants should be impressed with the necessity of respecting the body of the unconscious patient to the extent of refraining from using it as a means of support, either for themselves or for other objects, such as basins, pans and trays." The application of the electric pad to the back after operation is urged by Christopher.[3] Meyerding and Pollock[4] describe the condition and advise, concerning the treatment, as follows: "The backache which occurs as the result of overstretching of muscles and the relaxation of ligaments in the course of long anesthesia and which renders the immediate postoperative course a miserable experience, responds within a few days to applications of heat followed by sedative massage. A firmly applied binder, adhesive strapping or a lumbar sling are of great assistance in relieving the discomfort of which these patients complain." Da Costa,[5] however, has the following to say in regard to postoperative backache: "This is a very common and often a very distressing consequence of anesthesia. It is complained of soon after consciousness is regained, it may persist for several days, and is a not uncommon cause of wakefulness. It is usually greatly aggravated by turning, twisting, and by attempting to rise up from the bed. The pain is located in the lumbar and sacral regions and is often accompanied by rigidity of the lumbar muscles. Various explanations have been given for it. One view is that it is due to venal congestion. Another is that it results from congestion of the spinal cord. I believe that the explanation is that given by John Dunlop (New York Medical Journal—July 10, 1909), viz: 'The patients during the operation lay upon a flat table without support to the lumbar curve—consequently the sacroiliac synchrondroses were strained.'" The backache may be largely prevented by placing a small pillow so that it will support the lumbar curve during anesthesia. No mention, however, of postoperative backache is made in textbooks of surgery by Homans,[6] Christopher,[7] and Bickham.[8] Labat[9] and Dogliotti,[10] likewise, make no reference to the condition of postoperative backache in their contributions on anesthesia.

The following case is presented because the relief from postoperative backache afforded the patient by the local intra-

muscular infiltration with metycaine was prompt, complete and sustained:

CASE REPORT

F. L. S., a white male, eighty years of age, was first seen May 6, 1940, with the complaints of dysuria and frequency of urination of ten days' duration which had been becoming progressively worse. Rectal examination at that time revealed a moderately enlarged, tender prostate gland with the greatest swelling and tenderness localized in the left lateral lobe. Severe dysuria and extreme urgency developed the next morning and later during the afternoon an acute urinary retention occurred, with the bladder distended to the level of the umbilicus at 5:30 P.M. An attempt to introduce an indwelling catheter failed because of an obstruction encountered in the posterior urethra, where a peculiar "crackling egg shell" type of crepitus was experienced.

The patient was then referred to a urologist at Iowa Methodist Hospital, Des Moines, Iowa, who was finally able to insert an indwelling catheter after considerable instrumentation. A slow decompression of the bladder was then done. On May 13, 1940, cystoscopic examination revealed a large jagged "clinker" type of calculus imbedded in the posterior urethra. The calculus was removed in piecemeal fashion, and in order to give a badly traumatized posterior urethra a chance to heal, a suprapubic cystostomy was done. The patient then underwent a rather stormy convalescence marked by frequent bouts of abdominal distention and hiccups. About one week following the operation his postoperative course was further complicated, when he began to complain bitterly cf a severe persistent type of backache. The backache was always much worse at night, and of such intensity that it was impossible for him to obtain sufficient rest and sleep. The pain was partially controlled for the first four days by the hypodermic administration of codeine, but later morphine had to be given in increasing dosage to afford him adequate relief. Toward the end of his hospital stay, the morphine was gradually reduced, and then withheld completely as it was suspected that addiction to morphine had developed. Hypodermic injections of sterile water were then substituted for the morphine, and seconal and phenobarbital were given orally, but the patient complained constantly at night and occasionally during the daytime of severe back pain.

The patient was released from the hospital on June 12, 1940, at his own request and was returned to his hime. This decision was contrary to the advice of the consulting urologist. Backache, which then continued, was not very troublesome during the day. However, at night the pain became so severe that the patient was given ¼ gr. of morphine sulfate by hypodermic at bed time. Alcohol rubs, massage, hot water bottle applications and changes in position afforded him but slight and temporary relief. The patient's back muscles were examined every night and on each inspection, tender masses of spastic musculature extending bilaterally from the eighth thoracic vertebra to the fourth lumbar vertebra were palpated. The errector spinae, trapezii, and serrati posterior inferior muscles were affected to an equal degree on both sides.

On June 21, 1940, the backache became much worse and inspection of the back revealed rigid, exquisitely painful masses of muscle in the usual locations. Several hypodermic injections of morphine sulfate (¼ gr.) and dilaudid hydrochloride (¹⁄₂₀ gr.) gave the patient relief from pain for periods varying from four to eight hours. At 4 P.M. on June 23, 1940, 12 cc. of 2 per cent metycaine solution were injected through six intracutaneous metycaine wheals, directly into the rigid muscular masses. As a precautionary measure, however, one capsule containing 1½ gr. of seconal was given the patient fifteen minutes before the injection in order to neutralize any possible toxic effects due to metycaine. Partial relief from pain occurred in three minutes after the injection and complete relief experienced two minutes later lasted for a period of sixty-four and one-half hours. Recurrence of backache was then first noticed by the patient at 10:30 A.M. on June 26, 1940, and at 6:20 P.M. after the pain had become intense, 15 cc. of 2 per cent metycaine solution were injected through eight intracutaneous metycaine wheals, again directly into the mass of spastic musculature. One seconal capsule representing 1½ gr. of that drug was taken by the patient fifteen minutes prior to the injection. Complete relief from pain on this occasion was obtained in six minutes following the metycaine injection. The patient then enjoyed complete relief from pain for ninety-four hours

and forty minutes until 4 P.M. on June 30, 1940, when he complained of mild backache which was easily controlled by backrest, alcohol rubs and massage.

On July 1, 1940, he was moved to his daughter's home in the country for further convalescence. No recurrences of backache occurred during the next two months, after which he became ambulatory.

SUMMARY

1. An unusual case of severe and persistent postoperative backache in a white male eighty years of age is described.

2. Two per cent solution of metycaine was administered by local intramuscular infiltration on two occasions.

3. The patient was afforded complete and sustained relief from backache for periods of sixty-four and one-half and ninety-three and two-thirds hours, respectively.

REFERENCES

1. WARBASSE, J. P. and SMYTH, JR., C. M. Surgical Treatment. Vol. 1, p. 204. Philadelphia, 1937. W. B. Saunders Company.
2. CUTTING, R. A. Principles of Preoperative and Postoperative Treatment. P. 110–111. New York, 1932. Paul B. Hoeber, Inc.
3. CHRISTOPHER, F. Minor Surgery. P. 917. Philadelphia, 1934. W. B. Saunders Company.
4. MEYERDING, H. W. and POLLOCK, G. A. Backache: common causes and treatment with special reference to physical therapy. Arch. Phys. Therap., 21: 69–77, 1940.
5. DaCOSTA, J. C. Modern Surgery—General and Operative. P. 1135, 10th ed. rev. Philadelphia, 1931. W. B. Saunders Company.
6. HOMANS, J. A Textbook of Surgery. Springfield, 1931. Charles C. Thomas.
7. CHRISTOPHER, F. Textbook of Surgery. Philadelphia, 1936. W. B. Saunders Company.
8. BICKHAM, W. S. Operative Surgery. Vols. I–III. Philadelphia, 1924. W. B. Saunders Company.
9. LABAT, G. Regional Anesthesia—Its Technic and Clinical Application. 2d ed. Philadelphia, 1930. W. B. Saunders Company.
10. DOGLIOTTI, A. M. Anesthesia, Narcoses, Local, Regional and Spinal. Translated by Carlo S. Scuderi. Chicago, 1939. S. B. Debour.

ABDOMINAL NEUROFIBROMATOSIS*

CASE REPORT

JOSEPH SCHWARTZMAN, M.D.,
Associate Visiting in Pediatrics,
Metropolitan Hospital

LEO GROSSMAN, M.D.,
Clinical Assistant in Pediatrics,
Metropolitan Hospital

DAVID DRAGUTSKY, M.D.   AND
Clinical Assistant in Pediatrics,
Metropolitan Hospital

IRVING SHINER, M.D.
Resident in Surgery, Metropolitan
Hospital

NEW YORK, NEW YORK

WITH the ever increasing interest in new growths, which is rightfully merited, it is necessary to be on the alert for any condition that might come under this category, since the diagnosis very often determines the therapy and prognosis. When abdominally located, there are ever so many possibilities, one of the more infrequent ones being Von Recklinghausen's disease. In view of its relative rarity and yet its possibility in every case of neurofibromatosis, it was deemed worthy to re-emphasize this condition with the following case report:

CASE REPORT

C. C., a ten year old colored boy was admitted February 20, 1940, with a history of having epigastric pain which had begun one day before admission, following a heavy meal. He was given a cathartic and the pain subsided, but he had had no bowel movement for twenty-four hours. In addition, he gave a history of having a nodule on his head, which he said followed a fall out of a baby carriage during infancy, but it had not been present before.

His past history revealed that he was a normal delivery with normal postanatal development. He had had occasional sore throats and colds, scarlet fever at eight years and varicella at ten, while in 1938 he had a fracture of his leg with perfect recovery. The family history was essentially negative, the father having died of an unknown cause; but, so far as we could ascertain, there was no evidence in the family history of a similar lesion.

The physical examination revealed the following salient features: There was a ptosis of the left superior palpebrum with impaired vision in that eye. The cervical nodes bilaterally were enlarged, shotty and indurated. A subcutaneous tumor mass about the size of an egg was noted over the right side of the head, and subcutaneous nodules were found in the middorsal region, all of which were freely moveable. In addition, there were small, hard, freely moveable nodules over both upper extremities, especially on the extensor surfaces. There was also a hard mass palpable in the right lower quadrant of the abdomen and in the scrotum.

His blood Wassermann, blood chemistry, heterophile reaction, several urine examinations and throat culture were all negative. His mantoux test was positive. A blood count on February 20 revealed a hemoglobin of 68 per cent red blood count 2,850,000, white blood count 9,200, polymorphonuclears 52 per cent of which 4 were immature, lymphocytes 45 per cent and eosinophiles 3 per cent.

On February 29, a pyelogram showed that the ureters were displaced laterally below the fourth lumbar body, while the bladder showed extrinsic pressure which displaced it to the left and upward, giving the impression of an intra-abdominal mass probably retroperitoneal. A biopsy from a node on the right forearm revealed evidence of neurofibroma.

On March 7, a barium enema revealed no intrinsic pathological condition in the large bowel but showed extrinsic pressure on the rectal ampulla and distal sigmoid displacing this portion of the bowel to the right. X-rays of all the bones of the body were negative while another blood count on March 15 showed a hemoglobin of 70 per cent, red blood count

3,120,000, white blood count 8,760, polymorphonuclears 61 per cent, lymphocytes 36 per cent, eosinophiles 2 per cent and mononuclears 1 per cent.

extended well down, involving the sigmoid colon, the bladder wall, and was felt to extend well down on either side into the scrotum. It involved the peritoneum and the lateral wall

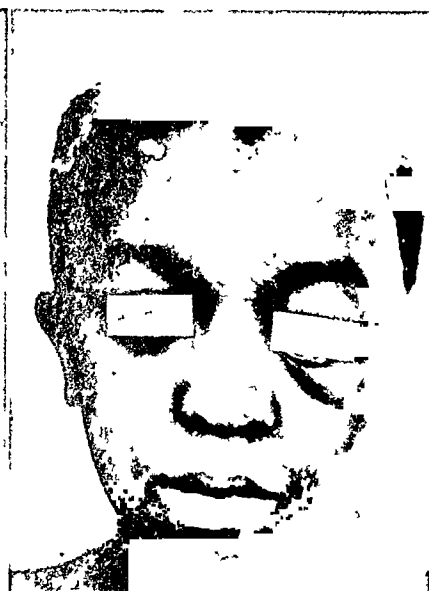FIG. 1. Encircled area is that of a neurofibroma on the right side of the head

FIG. 2. Edema and slight ptosis of the lids of left eye. Encircled areas indicate site of neurofibromas.

The abdominal mass continued to enlarge and in addition on March 25 edema of the left upper eyelid was noted, the left lower lid appeared to droop and on April 4 a slight swelling was noted in the infra-orbital region. A blood count at this time revealed a hemoglobin of 76 per cent, red blood count 3,600,000, white blood count 9,100, polymorphonucleus 60 per cent, lymphcytes 34 per cent, eosinophiles 3 per cent and mononuclears 3 per cent, which was a definite improvement over the previous results.

On April 9, a large nodular mass was noted involving the spermatic cord and epididymis while the original mass had extended to the umbilicus, was fixed to the abdominal wall and was irregular in contour. Rectal examination revealed a nodular mass infiltrating the anterior rectal wall.

On April 16, 1940, an exploratory laporotomy was performed. A right rectus incision was made and immediately upon opening the peritoneum, large masses were noted practically filling the abdominal cavity. The mass extended on the right side, well over the liver; and as it was traced downward, it was seen to extend across in front of the vertebral column and to infiltrate widely the mesentery. It

of the abdomen as well. The gross appearance of this mass was of a greyish, glass-like substance resembling fish meat. A large portion of the mass was removed for biopsy which, on section, preserved the glassy appearance.

Microscopically, there was noted proliferation of fibroblastic cells intermingled with fibrillar tissue and the diagnosis of Von Recklinghausen' disease was made.

The temperature, pulse, and respiration were normal throughout except on two occasions lasting two days each when the temperature rose to 102°F. because of acute pharyngitis and tonsillitis. The wound healed by primary intention and the patient was discharged on May 9, 1940.

DISCUSSION

Of the peripheral nerve tumors, there are two types,[1,2] the true neuroma which arises from or is composed of nerve fibers or cells; and the false type which arises from the nerve sheaths[3,4,5] of spinal or cranial nerves.

The latter [1,2] is the more common and within its classification is found Von Recklinghausen's disease, which is charac-

terized[1,2,3,5,6,7,8] by various sized, single or multiple, usually symmetrically located, nontender pigmented tumors; cutaneous

Histologically,[13,21,27,28,29] there is present proliferation of the perineurium, endoneurium and the ectodermal connec-

FIG. 3. Demarcated region on abdominal wall demonstrating the size of the palpable abdominal mass. The vertical lines show the site of the incision. A neurofibroma of the right forearm is also noted.

dysplasia in the form of yellow or brown pigmentations with the general skin often being darker than that of other members in the family; and an associated disturbance in growth[9,10] and mentality[3,5,7,11,12,13] in some cases.

The tumors are mostly congenital[3,12] and appear at birth or very soon thereafter and may either remain unchanged until activated[5,7,8,14,15] at puberty or later, or during pregnancy or menopause, or may increase in size and number early and rapidly. Removal[12,14,15,16] is often followed by recurrences; and cystic, myxoid or malignant degeneration,[3,13] the latter especially in ganglioneurofibromas,[17] has been observed in 12 to 14 per cent of the cases.[8]

Their distribution[5,6,8,12,18] is by no means confined to the skin and subcutaneous tissue but may be wide and variable, having been encountered in the abdominal,[6,12,13,14,15,18,19,2] cranial,[6,21] spinal,[4,22] and thoracic cavities,[8,15,20,23,24] as well as the bony structures[6,7,10,25] and in association with the organs within and about these areas. Pressure symptoms may arise and in the abdominal type[12,26] may result in all clinical varieties of obstruction, hemorrhage,[14] and perforation. At times, melanotic placques[2] in the epidermis may cause paralysis of isolated nerves.

tive tissue (Schwann's sheath) so that the tumor appears to be composed mostly of small sized, mature cells from connective tissue which surround nerve fibers that are often swollen and irregular, and this gives a typical palisade formation and whorling fibrous structure which is characteristic of neurofibroma.

Etiologically, heredity[1,2,3,4,5,7,30,31] has been demonstrated in 18 to 74 per cent of the cases.[8,32]

Among the conceptions[4] offered as to its origin have been: (1) That they are malformations and groups of aberrant cells; (2) that an endocrine disturbance[9,33,34,35] is the basis since many cases[5] have been associated with growth changes; (3) that it is due to an anomaly[3] of development of the nervous system in early fetal life.

The condition[3,8] is not sex linked and tends to recur in successive generations of involved families and, therefore, some[5,7,8,31,36] believe it is inherited as a Mendelian dominant character.

Regarding its treatment when located within any of the body cavities or organs, surgery and deep irradiation[16] have been the methods employed, with some reporting good results,[4,5,8,12,14,16,18,19,20,24] others mortalities,[6,13,21] while some[16] have noted recurrences following removal of the tu-

mors. However, in these cases some active form of therapy is necessary for the relief of symptoms and surgery is the method of choice with follow-up by irradiation.

SUMMARY AND CONCLUSIONS

1. A case of massive abdominal neurofibromatosis in a colored child is reported.

2. In view of its hereditary nature, there should be a constant lookout for the condition in all blood relations of any affected family.

3. In every dermal case, the threat of visceral involvement is a constant potentiality.

4. Therefore, if any visceral symptoms coincide with cutaneous manifestations, the assumption should be that there is an analogous tumor involvement of the viscera until proved otherwise.

REFERENCES

1. POLLACK, L. J. Cecil's Textbook of Medicine. 4th ed., p. 1439. Philadelphia, 1936. W. B. Saunders Co.
2. WOLTMAN, H. W. Tice's Practice of Medicine. Vol. 9, p. 361. Hagerstown, Md., 1925. W. F. Prior Co.
3. FORD, F. Brennermann's Practice of Pediatrics. Vol. 4. Hagerstown, Md., 1937. W. F. Prior Co.
4. ALESSI, D. Policlinico, 46: 2043, 1939.
5. LEVIN, D. L. and BEHRMAN, H. T. Arch. Dermat. & Syph., 41: 480, 1940.
6. GOLM, G. and CHRISTELLER, E. Med. Klin., 22: 1188, 1926.
7. PREISER, S. and DAVENPORT, C. Am. J. M. Sc., 156: 507, 1918.
8. JONES, R., JR., and HART, D. Ann. Surg., 110: 916, 1939.
9. GORDON, M. B. Endocrinology, 13: 553, 1929.
10. LEHMAN, E. and BROOKS, B. Surg., Gynec. & Obst., 38: 587, 1924.
11. CHARPENTIER, J. Encéphale, 5: 460, 1910.
12. JUARISTI, V. Rev. de cir. de Barcelona, 11: 133, 1936.
13. JENTZER, A. and FATZER, H. Schweiz. med. Wchnschr., 67: 569, 1937.
14. BARTON, A. D. and KEITH, I. J. Coll. Surg. Australasia, 3: 397–402, 1931.
15. SERRE, R. Contribution to the Study of the Visceral Localizations of Recklinghausen's Disease. Thesis, Paris, 1923.
16. ADSON, A. W., MOERSCH, F. P. and KERNOHAN, J. W. Arch. Neurol. & Psychiat., 41: 535, 1939.
17. LEWIS, D. and GESCHICKTER, C. F. Arch. Surg., 28: 16, 1934.
18. HOEY, T. Brit. M. J., 2: 490, 1928.
19. WOLFF, P. Schweiz. med. Wchnschr., 66: 379, 1936.
20. WRIGHT, F. H. and PAIGE, B. H. J. Pediat., 14: 137, 1939.
21. SCOTT, P. Proc. Roy. Soc. Med., 31: 1417, 1938.
22. GORDON, A. Arch. Dermat. & Syph., 37: 983–986, 1938.
23. DYGGVL, L. P. Finska läk.-sällsk. handl., 79: 133, 1936.
24. MAKKAS, M. Bruns' Beitr. z. klin. Chir., 159: 276, 1934.
25. STAHNKE, E. Deutsche Ztschr. f. Chir., 168: 6–18, 1922.
26. SMITH, F. R. Am. J. Cancer, 15: 859–862, 1931.
27. RHOADS, C. P. and VAN WAGENEN, W. P. Am. J. Path., 4: 145–152, 1928.
28. PENFIELD, W. and YOUNG, A. W. Arch. Neurol. & Psychiat., 23: 320, 1930.
29. PENFIELD, W. Arch. Neurol. & Psychiat., 27: 1298–1308, 1932.
30. BUCY, P. C. Tice's Practice of Medicine. Vol. 9, p. 569, 1925.
31. GARDNER, W. J. and FRAZIER, C. H. Arch. Neurol & Psychiat., 23: 266, 1930.
32. SHARPE, J. C. and YOUNG, R. H. Arch. Int. Med., 59: 299–328, 1937.
33. TUCKER, B. Arch. Neurol. & Psychiat., 11: 308–320, 1924.
34. ELLIOTT, C. A. and BEIFELD, A. F. J. A. M. A., 63: 1358–1362, 1914.
35. JACKSON, A. H. J. Nerv. & Ment. Dis., 78: 581–596, 1933.
36. FRETS, G. P. Genetica, 11: 347–366, 1928–1929.

THE USE OF A STERILE RUBBER GLOVE AS A COVERING FOR THE DURAL DEFECT RESULTING FROM THE REMOVAL OF A SPINAL CORD TUMOR

CASE REPORT

MAJOR WALTER G. HAYNES, M.C.

Chief of the Neurosurgical Section, Lovell General Hospital

FORT DEVENS, MASSACHUSETTS

THE following case is reported to present a method of repairing dural defects which result from either the necessary or the traumatic resection of a part of the dura covering the brain or spinal cord. It has long been a neurosurgical custom to replace resected dura with a sheet of Cargile membrane or animal membrane, with the idea that this preparation will afford some means of protection while the dura is rapidly being regenerated beneath it. The membrane is rarely sutured to the surrounding dural edges but is simply placed over the defect. It is difficult to secure these membranes and, since this is particularly true in the armed services where supplies are of necessity limited, the procedure herein to be described may prove of value to surgeons in combat areas who have occasion to repair dural defects but who do not have available the finer equipment and materials usually employed.

CASE REPORT

A white soldier, twenty-three years old, was admitted to Lovell General Hospital on January 19, 1942. He had felt well until six months prior to his admission to a Station Hospital. He began to notice that his legs were becoming weak and found that he could not jump a hurdle higher than three feet, whereas previously he had shown athletic ability. This weakness persisted but he thought little of it. Two months later, when on maneuvers, he noted that he was unable to perform his duties as well as did the other soldiers. His legs seemed weak and stiff. He was hospitalized at a Station Hospital for approximately one month and was then transferred with a diagnosis of multiple sclerosis to another Station Hospital. Meanwhile the weakness and stiffness of the lower extremities became progressively worse. There were occasional fibrillary twitches in the muscles of the legs, as well as a tingling sensation over the entire lower half of the body and legs. He staggered when he walked and felt as though he were walking on pillows. At no time did he experience pain. He had difficulty in starting his urinary stream and was constipated. All of these symptoms progressed so that at the time of his admission to Lovell General Hospital he was unable to stand or walk, and any slight change in position of his lower extremities produced an extreme, sustained ankle clonus. However, he still had some control over his bladder and bowel functions and he did not complain of pain.

Neurologic examination at this time revealed normal function of the cerebrum and cerebellum and of the cranial nerves. Examination of the upper extremities was negative, but examination of the lower extremities revealed the muscles to be weak, almost completely paralyzed and hypertonic. There was anesthesia extending from the anterior costal margin downward over the trunk and over both legs. There was loss of sensation of pain and temperature, although that of light touch was preserved. Vibratory sense, as well as position sense and deep sensibility, was lost from the iliac crest downward bilaterally. The reflexes were exaggerated. The Babinski and Rossolimo signs were positive. There was a sustained ankle clonus on both sides. Careful roentgenographic studies of this area were negative, but spinal puncture revealed a complete subarachnoid block with an increased spinal fluid protein of 85 mg. per cent. There was a positive Beevor's sign in that the umbilicus moved upward upon flexion of the

body. The abdominal and cremasteric reflexes were absent. The remainder of the study of the patient was negative. A diagnosis was made of meningioma at the level of the fifth dorsal vertebra.

A dorsal laminectomy was performed on January 26, 1942. The operative report follows: "Under local anesthesia, the usual sub-periosteal approach to the lamina of the fifth, sixth and seventh dorsal vertebrae was carried out. The spinous process and the uppermost portion of the lamina of the fifth dorsal vertebra were removed and it could be seen that the cord was pulsating freely at this level. It was assumed that the block must exist caudad so I then began to remove the lower portion of the lamina of the fifth dorsal vertebra and all of the lamina of the sixth dorsal vertebra. Immediately upon reaching the inferior portion of the fifth dorsal vertebra, a firm reddish mass could be seen, which was extradural and compressing the dura to the right. This was followed down, necessitating the removal of all of the laminae of the sixth dorsal vertebra and a few bites of the upper portion of the seventh dorsal vertebra. The tumor was then exposed in its entirety. It consisted of an envelope which covered the entire posterior portion of the dura and extended laterally on the left side to almost the midline anteriorly. This tumor, presumably a meningioma, was 1 cm. thick and approximately 7 cm. long and was of a firm consistency. The area was cleaned up, the epidural fat removed, and I began the attempt to remove the tumor. This was accomplished by wiping away the discrete mass from the lower and superior portions with cotton patties, using silken threads as retractors. It was then found that the posterior, lateral and anterior portions of the tumor combined and extended through the intervertebral foramen, encompassing the nerve root at this level and forming a dumb-bell on the outside of the bone. This tumor was removed, separating it from the dura and sacrificing the nerve root. The portion that was outside the bone was curetted out with a pituitary spoon and the few cells that were remaining outside of the bone were coagulated with the Bovie unit. Then because of the fact that the tumor had arisen from the dura at this point, the dura being involved for a distance of 4 cm., I opened the dura. This was done with no undue difficulty and it was found that a tumor, almost as large as that outside the dura, was growing inside and severely compressing the cord at this point. This tumor also was wiped away, necessitating the sacrifice of the posterior root. It was also attached to the dura so that it became necessary for me to sacrifice a length of dura 4 cm. long and 2 cm. wide. The entire procedure had been very vascular, as is usually the case when dealing with a meningioma. Hemostasis was obtained by means of the Bovie unit and muscle stamps. It then became necessary for me to replace the sacrificed dura, which is usually done by using a sheet of animal membrane, not available in this hospital. Accordingly, I used the back of a sterile rubber glove and repaired the dural defect with this thin sheet of rubber. The wound was closed in the usual fashion. The patient's condition at the termination of this operation was good."

The postoperative course was uneventful. Upon his return to the ward, the patient noticed an immediate improvement in the function of his lower extremities. The ankle clonus had disappeared, as had the positive Rossolimo and Babinski signs. His condition improved rapidly and on the eighth day, when the sutures were removed, there was no anesthesia present. There was still some posterior column involvement, a condition to be expected since the greater portion of the tumor lay on the posterior column. The spasticity and increased reflexes had also disappeared. At the end of two weeks he was walking. He was somewhat ataxic but this condition has gradually disappeared. He is walking normally and has only a slight diminution of vibratory sense below the iliac crest. The wound remained clean and healed well, with no indication of any reaction, there being only a fine linear scar resulting from the incision. During the four months following the operation, repeated white blood cell counts and sedimentation rates were studied, as well as roentgenograms of the area involved. The white blood cell counts and the sedimentation rates have all been normal and roentgenograms have shown no sign of an inflammatory process. Spinal puncture, performed a month after the operation, revealed no evidence of a subarachnoid block. There were no cells in the spinal fluid. The microscopic sections of the tumor proved it to be a meningioma.

SUMMARY

1. An operation is described during the course of which a large area of dura had to be sacrificed to facilitate the removal of a large, double dumb-bell type of spinal cord tumor. This dura was replaced with rubber from the back of a sterile rubber glove and the patient had an uneventful convalescence with no evidence of inflammatory reaction.

2. The method of repair of dural defects described was suggested by the original work of Giangrasso,[1] who covered dural defects in animals with rubber from discarded gloves and who found that dural regeneration was rapid and that restoration of dural continuity was achieved without the formation of adhesions.

3. This method may be of particular value in the armed forces where Cargile membrane or other animal membranes are difficult to obtain.

REFERENCE

1. GIANGRASSO, G. Use of rubber glove to cover dural defects in dogs. Ann. ital. di chir., 19: 431–435, 1940.

DYSPHAGIA may be defined as an embarrassment of swallowing due either to painful, nervous, or anatomical interference with this function. The cause of painful deglutition may be present in the mouth, pharynx, hypopharynx, or esophagus itself.
From "Symptoms in Diagnosis" by Jonathan Campbell Meakins (Little, Brown and Company).

ACUTE HEMATOGENOUS STAPHYLOCOCCIC OSTEOMYELITIS OF FEMUR

SUCCESSFUL TREATMENT WITH SULFATHIAZOLE WITHOUT OPERATION

L. M. Rankin, m.d. AND Sherman A. Eger, m.d.

Chief of Service, Delaware County Hospital Assistant Surgeon, Delaware County Hospital

UPPER DARBY, PENNSYLVANIA

IN December 1941, Hoyt, Davis and VanBuren reported the first cases of acute hematogenous osteomyelitis treated with sulfathiazole without operation. The results in their eight cases were so encouraging that we decided to try out this method of therapy.

CASE REPORT

On December 13, 1941, a white boy, age fourteen, was admitted to the surgical service at the Delaware County Hospital with a diagnosis of acute osteomyelitis of the right femur. One week prior to admission he had received a hard bump on the right thigh while playing football, from which he had apparently completely recovered during the next two or three days. Twelve hours before admission the patient developed pain along his entire right thigh, which was continuous and progressive. Six hours later the pain was quite intense and was accompanied by chills, fever and headache. Tonsils and adenoids had been removed one year before. There had been no other operations and no serious illnesses.

Physical examination revealed a well developed boy with signs of toxemia but well orientated and co-operative. No focus of infection was demonstrable. Pathologic findings were limited to the lower one-third of the right thigh where there was marked tenderness and slight swelling, especially on the anterolateral aspect, near the epiphysis. There was no redness, induration or fluctuation. The knee joint did not appear involved, was most comfortable in a semiflexed position, and any attempt to change this position produced marked pain just above it. Temperature was 102²°F., pulse 118, respiration 22, hemoglobin 104 per cent, red blood cells 4,900,000, white blood cells 17,200, polymorphonuclears 71 per cent, stabs 12 per cent, segmented 59 per cent. Urine was normal.

An initial dose of sulfathiazole, Gm. 2.0, was given by mouth followed every four hours by Gm. 1.0. In addition, oral fluids were freely taken and well tolerated. Hot saline dressings were applied locally and the leg immobilized. Three hours after admission temperature rose to 104²°F. and pulse 132 and continued at about this level for forty-eight hours, following which a gradual decline occurred associated with an improvement of symptoms. Sulfathiazole blood level was only 2.0 mg. per 100 cc. at this time. Blood culture on the second day showed staphylococcus in four days. From the sixth to the ninth day the temperature and pulse stabilized at about 100 and 110, respectively, and at this point the dose of sulfathiazole was increased to Gm. 2.0 every four hours. A gradual improvement followed so that by the fourteenth day his temperature and pulse were normal and remained so. A blood transfusion of 250 cc. of whole blood was given on the fourteenth day because of a mild secondary anemia and for a general supportive measure. Because of the disappearance of the signs and symptoms sulfathiazole was discontinued on the eighteenth day. The patient was out of bed on the nineteenth, walking on the twenty-first and discharged on the twenty-fourth day. X-ray examinations of the femur three days and thirteen days after onset of symptoms were negative. Since discharge from the hospital there has been no recurrence of symptoms, his physical activities have been unrestricted, and an x-ray taken March 12, 1942, three months after onset of symptoms, was negative.

The reasons for reporting this case are (1) It presents a new method of therapy; (2) by reporting individual cases a large series from different surgeons can be gathered together and properly evaluated; (3) this is a typical case clinically; (4) complete recovery was secured without operation or any subsequent indication for

operation; (5) there was a comparatively short period of hospitalization with no deformity resulting; and (6) it is important from a military standpoint.

CONCLUSIONS

1. No attempt should be made to treat this disease in the home.

2. The patient should be hospitalized and treatment instituted twenty-four hours after onset.

3. The earlier sulfathiazole treatment is begun, the better should be the result, in extent of bone destruction, period of hospitalization, disability and mortality.

4. Low blood sulfathiazole level was due to forcing of fluids. When forcing of fluids is necessary, the dosage of sulfathiazole should be increased rather than decrease the fluid intake.

5. Immobilization of affected part decreases pain and avoids irritation of inflammatory area.

6. Several supportive measures should not be neglected, such as fluids and transfusions, caloric intake—vitamins, proteins, minerals, etc.

REFERENCE

Hoyt, Davis, and VanBuren. Acute hematogenous staphylococcic osteomyelitis. J. A. M. A., 117: 2043-2050, 1941.

Massage and passive motion have a distinct use in preserving muscle tone and maintaining blood and lymph circulation. However, both of these measures may be used with care and judgment. A good working rule is that which says passive motion that causes pain is harmful.

From "A Manual of the Treatment of Fractures" by John A. Caldwell (Charles C. Thomas).

SPONTANEOUS RUPTURE OF THE UTERUS IN THE FOURTH MONTH OF GESTATION

CASE REPORT

J. M. ALEXANDER, M.D.

ARGOS, INDIANA

THE following is a case report of spontaneous rupture of the uterus in the fourth month of gestation:

Mrs. J. H. was first seen November 27, 1941. Her chief complaint was sterility. She had been married two years and wanted children badly, but had been unable to conceive. The patient was an obese, white female, nineteen years of age. After an examination she was found to be hypothyroid and was put on thyroid medication. During the first three weeks on thyroid medication the patient lost fifteen pounds in weight. Two months after her first visit she missed her first menstrual period. Two months after the onset of her amenorrhea a pelvic examination revealed an enlarged uterus.

After becoming pregnant the patient experienced no particular symptoms except a moderate morning sickness. On the morning of May 12, 1942, I was called to see her and found she was having uterine contractions which I interpreted as a threatened abortion. There was no vaginal bleeding. The patient was given an injection of a barbiturate. I went to my office to get some progesterone and vitamin E and when I returned to the patient within an hour found her in deep shock. She was pallid, pulseless, cold, sweating, and only semiconscious. She was given coramine, hot water bottles were put to the body, and the patient was taken to the Kelly Hospital in Argos, Indiana, where she was seen in consultation with Dr. F. H. Kelly. The patient was moribund when she entered the hospital and expired in about thirty minutes after admission, despite the use of artificial respiration, oxygen and stimulants. Expiration took place about four hours after the onset of symptoms.

The clinical picture was obviously due to intraperitoneal hemorrhage, but it was impossible to operate without controlling the shock. We did not have time to type the patient for a transfusion, and a transfusion would have been very difficult because of the obesity, very tiny veins and their collapse due to shock. It was impossible to determine the source of the hemorrhage because I was sure it was a uterine and not an ectopic pregnancy.

On opening the abdomen at autopsy the peritoneal cavity was found to be filled with liquid, clotted and semiclotted blood. The blood was wiped away, and the placenta was encountered lying about the legs of the fetus. The umbilical cord was twisted around the legs of the fetus. The fetal head still lay within the uterus. The fetal neck was emerging from an opening in the superior aspect of the fundus. The fetus, a female, was 15 cm. long. The uterus was contracted down so that the cavity would just contain the fetal head.

The mother on my first visit denied a fall or any other injury, and the couple wanted a child so badly I am sure no attempt at an abortion had been made. Therefore, I am constrained to believe this was a case of spontaneous rupture of the uterus. The patient had never had a previous abdominal operation.

The only explanation I can think of is that there was a congenital hypoplasia of the myometrium, and as the uterus enlarged a bulging occurred just as an inner tube will bulge through a weak place in a tire. Contractions were initiated, and the fetus was forced through the weak place, rupturing the uterus, and causing death from an intraperitoneal hemorrhage. There was never any vaginal bleeding. No histological study of the uterus was made.

New Instruments

A WIRE SPLINT FOR WRIST

MAJOR DAVID SLOANE, M.C., U.S.A.

NEW YORK, NEW YORK

A WRIST splint which is simple, light, inexpensive and easily made in a matter of minutes from readily available material is herein described.

distance from the midpalm to about 2 inches short of the fold of the elbow. The ends of the wire are looped over in opposite planes and held in place with adhesive.
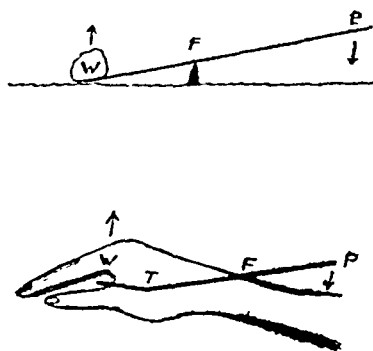
FIG. 1. Lever principle applied to wire splint: P, power; W, weight to be lifted; F, fulcrum. Arrows denote direction of applied and resultant forces. The angle at point T enables us to increase the amount of dorsiflexion to the desired amount.
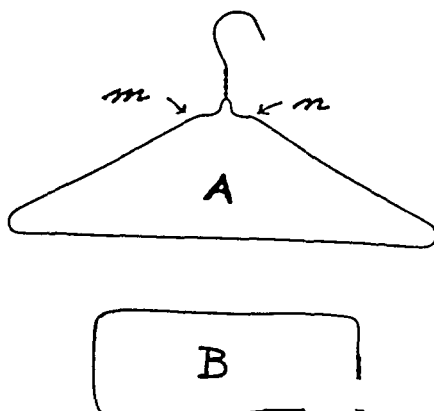
FIG. 2. A, standard type of wire coat-hanger. Cut wire at points m and n, and discard the hook section. B, Rectangle of wire held together by looping ends in opposite planes and fastening with adhesive. Excess of wire may be cut off and discarded.

Based on the mechanical principle of a first class lever, the splint pushes the wrist into dorsiflexion. (Fig. 1.)

Take an ordinary wire coat-hanger (Fig. 2A), cut away the entire hook part and discard it and straighten out the remaining wire. This will yield a wire rod $\frac{3}{32}$ of an inch in diameter and about 30 inches long. Bend this wire rod into a rectangle. (Fig. 2B.) The width of the rectangle is a trifle larger than the diameter of the clenched fist and its length is the

The distal cross-bar is padded with any soft material. The patient then grasps this padded handle-bar and the rectangle rests on the dorsum of the forearm. At the level of the radial styloid the splint is bent upward approximately equivalent to the desired angle of dorsiflexion. (Fig. 3B.) Trial and experience will determine the angle of upward tilt. Cover the proximal two-thirds of the splint with an apron of double adhesive or any similar firm material. (Fig. 3A.) As pressure is applied to the

apron the padded handle-bar forces the wrist into a cock-up position. The fulcrum of leverage is at the free edge of the apron.

adhesive, covered by a firm bandage, keeps the splint in position. (Figs. 4 and 5.)

The resiliency of the wire is such that a
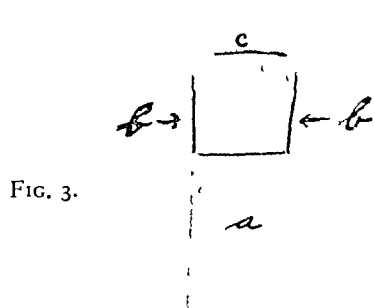
FIG. 3.

FIG. 4.

FIG. 5.

FIG. 3. Completed splint. a, adhesive apron, b, angulation for increased dorsi-flexion, c, handle bar.
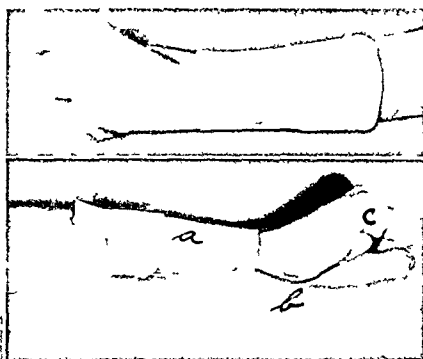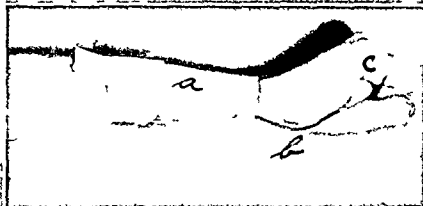
FIG. 4. Top view of splint in use.
FIG. 5. Side view of splint in use. For simplification the forearm bandage has been left off: a, apron; b, angulation point; c, handle bar.

The proximal cross-bar is bent in a curve to conform to the rotundity of the dorsum of the forearm. This will enable the splint to lie flat without shifting. A broad strip of

certain amount of voluntary flexion is possible but the wrist immediately springs back into extension. Should more rigidity be desired, heavier wire may be used.

A NEW MECHANICAL FRACTURE SPREADER

F. T. BRENNER, M.D.

Orthopedist to St. Mary's Hospital

QUINCY, ILLINOIS

TRACTION and fixation are the two necessary principles for proper reduction of every fracture. Temporary traction alone does no good unless the the way of plaster cast application. The instrument here illustrated, and as shown further in the text, covers all of these requirements.
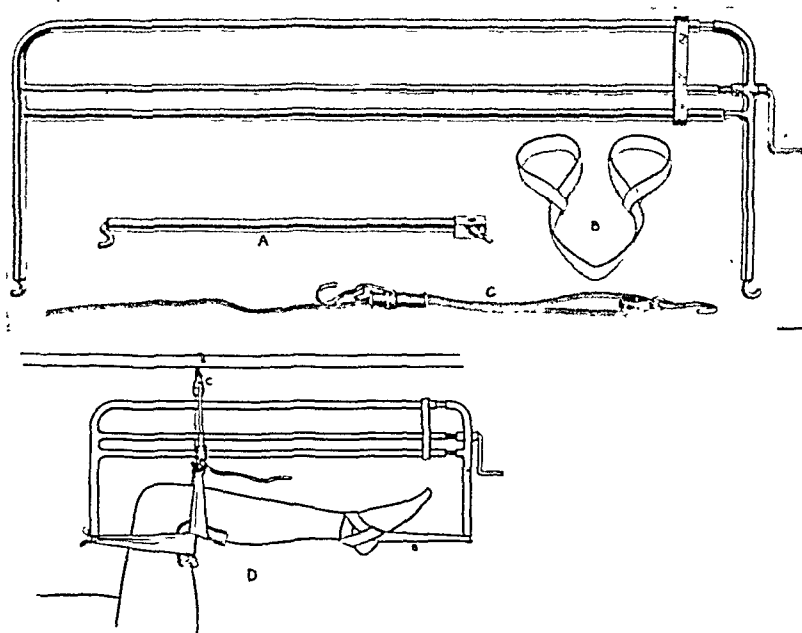
FIG. 1. Fracture reduction apparatus and accessories. The double arm with the traction screw represents the complete instrument as shown in insert. A, the extension piece to be used when a wider spread is desired; B, Brenner ankle or wrist hitch. The two loops slip over ankle and are drawn taut. C, Brenner overhead pole hitch with automatic lock and pulley to suspend leg while under reduction or cast. D, Insert,—showing complete action on leg fractures.

fragments can be held firmly in proper position. Also it is necessary that the associated muscles be in relaxation while traction is on. For accurate adjustment we must be able to view the position of fragments with the fluoroscope at any angle without disturbing their allignment while reduction is being done.

Repeated x-ray pictures, and then a little more adjustment is too time-consuming and expensive. Therefore, your instrument must not be in the way of the fluoroscope. The instrument must also be entirely out of

The principle is simple: There are two arms that are separated by screw action, guided by trombone action of the two sleeves. The hitch or cord makes the pull from the arm hooks. It is made with an easily detachable extension piece permitting a spread of 6½ feet or more. Only seldom is it necessary to use this extension, that is, when it is desired to make traction on a tall adult with one hitch in the groin and the other from the ankle.

Ordinarily one will prefer to make traction with the knee flexed in order to relax

the muscles better, either for a femoral fracture or a fracture of the lower leg. With the knee flexed the extension piece is not needed.

For a fracture of tibia or fibula a heavy piece of felt is placed under the knee and the flexed leg is suspended by this from an overhead pole by pulley cord which automatically locks in any position.

If hitch traction is desired, the leg is suspended as before. Another felt pad is placed under the leg just above the knee, and this hitch is hooked to one arm of the tractor. The other tractor arm is hooked to Brenner's ankle hitch. A few turns of the traction screw—measured by the scale— brings the fragments in line.

The fracture spreader can be swung around, up or over, or to one side, perfectly out of the way of the fluoroscope for any view. The splints or cast can then be applied with no interference from the instrument. In order to make traction with the knee in extension, one hitch is placed in the groin and the other on the ankle. Now the extra extension piece is used, quickly attached and firmly held by a single friction lock.

Forearm Fracture. A Brenner hitch is placed on wrist and a felt pad is placed above the elbow with the elbow flexed at 90 degrees; or the arm is extended with one hitch in the axilla and the other on the wrist.

Humerus. One hitch is placed in the axilla and the other at the bend of the elbow, or pin hitch.

Hip Dislocation. A felt pad is placed below the knee with the knee sharply flexed. The other hitch is applied in front of the groin with the hook behind the buttocks. Then while traction is on the flexed leg can be manipulated similar to the Kocher shoulder dislocation technic. The spreader* swings around with the leg to any position without lessening the pull. Whenever bone pin traction is desired, a double loop can be made with a piece of strong cord; hook a loop over each end of the pin. The double loop is then attached to each spreader hook and traction made as before. The cord remains on the pin and is included in the cast. This is the ideal method by far.

Brenner suspension pole hitch is a great convenience to suspend the leg in any desired degree of flexion, either while reducing the fracture or while putting on the cast. The large hook hangs from an overhead pole. The rope from the pulley has an automatic lock attachment. A simple pull suspends the leg and holds it there. There is no tiring effort or shifting of position as when held by an assistant.

The *Brenner ankle or wrist hitch* is made of strong two-inch web strap. It automatically adjusts itself to an infant or adult wrist or ankle. The strap lies flat and does not cut by wrinkles or creases. It will not slip, is easy to put on and is easy to remove.

* This instrument is light, easily portable, adapted to any long bone fracture and after adjustment is entirely mechanical. It swings out of the way of fluroscope and cast without disturbing the fracture allignment and pull. It is suitable for either hitch or bone pin traction. Scale to measure traction.

A TUBAL INSUFFLATION METHOD

HARRY S. FIST, M.D.

Diplomate, American Board of Obstetrics and Gynecology

LOS ANGELES, CALIFORNIA

THE Rubin test for patency of the fallopian tubes is a method of passing gas through the cervix, fundus and tubes, into the abdomen.

now available is expensive and more or less complicated.

The Dickinson modification of the Rubin insufflation apparatus is simple and effi-
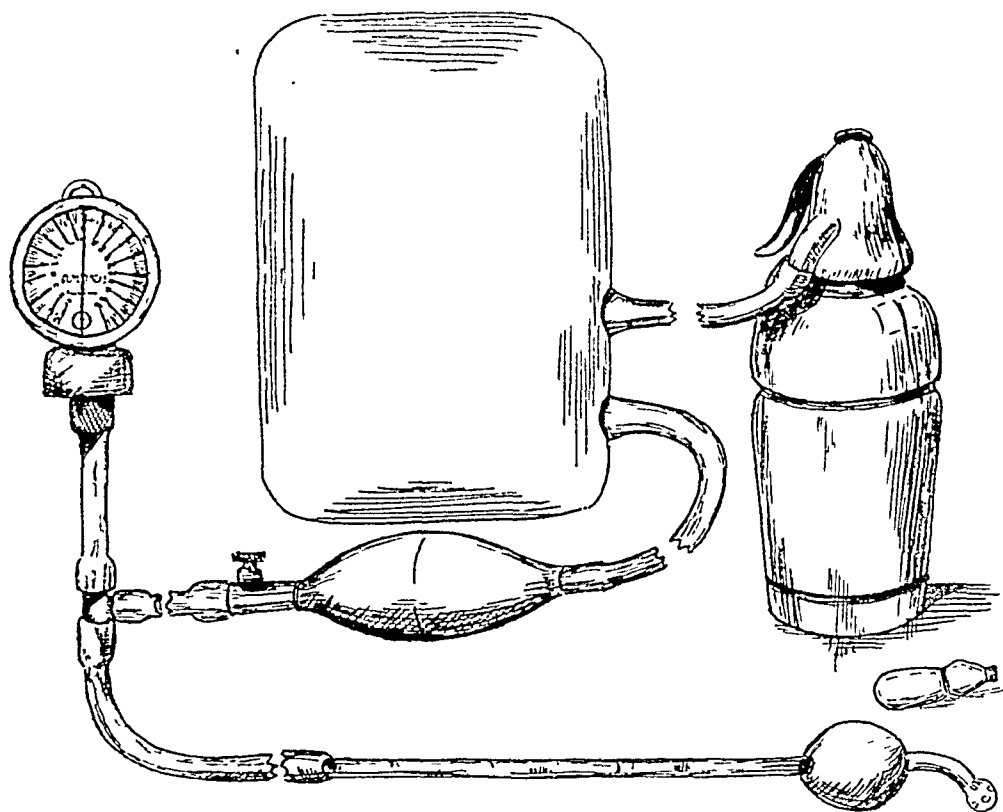
FIG. 1. Carbon dioxide released from the dry syphon partly inflates the rubber bag. With the hand bulb the operator pumps the carbon dioxide from the bag into the uterine cannula as he checks the pressure on the manometer.

Experience has demonstrated that "room air" has several characteristics which make its use objectionable. Air contains nitrogen, which is absorbed slowly. The discomfort following its use is, therefore, somewhat long-lasting and air is theoretically more apt to cause gas embolism.

Carbon dioxide, being more rapidly absorbed, shortens discomfort time and lessens the possibility of gas embolism; however, the carbon dioxide apparatus as

cient.* It consists of a rubber bulb with a thumb operated release, a pressure gauge and a cervical cannula, all connected with rubber tubing. It lends itself well to office use, but uses ordinary room air.

The author's instrument (Fig. 1), utilizes the cannula, pressure gauge and rubber bulb described by Dickinson. In addition to these, facilities are provided for supply-

* Robert L. Dickinson. Nelson Looseleaf Surgery, p. 751, 1929.

ing carbon dioxide under low pressure instead of air. This gas may be pumped into the uterus as desired, using the rubber bulb in the same manner as for air. An ordinary household syphon supplies carbon dioxide from a small cartridge. The arm band from a blood pressure instrument provides a reservoir for the gas. One of the rubber tubes is attached to the syphon. The other tube is attached to the rubber bulb by means of a metal connection which has been soldered to the intake valve of the bulb. The pressure gauge is attached to one side of a "T" tube, and indicates the pressure caused by the bulb as gas is pumped to the uterus.

METHOD OF USE

The dry syphon is filled with carbon dioxide from a new gas cartridge. The syphon handle is pressed for a moment to remove the air from the instrument and to fill with carbon dioxide. The sterile cannula is inserted into the cleansed cervix and the syphon handle is pressed to deliver carbon dioxide into the rubber bag. Then, with the bulb, the carbon dioxide is pumped into the uterus slowly, while the pressure gauge is being watched. Thus, carbon dioxide is supplied instead of air.

This method avoids the use of gas pressure tanks and complicated valves, with danger of sudden pressure increase. The carbon dioxide is released into the rubber bag and there stored at low pressure until pumped by the hand bulb into the uterine cavity.

Correct pressure control is checked by means of the gauge and the hand bulb is used precisely as is done when the air instrument is used.

Bookshelf Browsing

SURGEON JOHN JONES, U. S. ARMY

FATHER OF AMERICAN SURGERY AND AUTHOR OF AMERICA'S FIRST SURGICAL BOOK

Colonel·Edgar Erskine Hume, m.c.

United States Army

CARLISLE BARRACKS, PENNSYLVANIA

AMONG the great things which medical officers of the United States Army have contributed to science are three books, each a milestone in its particular field. They are (a) the first American book on surgery, by Surgeon John Jones (1729–1791) which was published in 1775; (b) the first American pharmacopœia, by Physician General William Brown (1748–1792) which appeared in 1778; and (c) the first American textbook on bacteriology by Surgeon General George Miller Sternberg (1838–1915), which came out in 1884. The first American publication on vaccination, by Surgeon Benjamin Waterhouse (1754–1846) appeared in 1799, and might be added to this list, though it was a short communication and not a book. One must also, in consideration of priorities, give paramount place to the *Index Catalogue* of the Army Medical Library, America's greatest contribution to bibliography and the greatest catalogue of all time.

The earliest of these books is the monograph of Surgeon John Jones. This work and its author are not as well known as they deserve to be. The book is at once the first medical book by an American, the first American work on surgery, and the first work on hygiene that our country produced. Hygiene is contained in an Appendix. "At the commencement of the Revolutionary War," said Billings in his *A Century of American Medicine* (1876),

"we had only one book by an American author, three reprints, and about twenty pamphlets." This unique book was Jones's monograph.

Jones's book bears the lengthy title usual in the eighteenth century: *Plain Concise Practical Remarks on the Treatment of Wounds and Fractures; To which is Added a Short Appendix on Camp and Military Hospitals; Principally Designed for the Use of Young Military Surgeons in North America.* New York, 1775, 92 pages.

The author, an Army medical officer, was one of the great American surgeons of the period. He was a founder and the first Vice-President of the College of Physicians of Philadelphia, a founder and Professor of Surgery at King's College (now Columbia University), and a founder and the designer of the New York Hospital. When we add these to his having written the first American medical book, we cannot help but recognize Jones as one of the great men of his day.

BIRTH

John Jones was the son of Dr. Evan Jones and Mary Stephenson. His grandfather was Dr. Edward Jones, whose wife, Mary, was the eldest daughter of Dr. Thomas Wynne, one of William Penn's fellow passengers in the *Welcome*. John Jones was born at Jamaica, Long Island, in 1729. All four of his grandparents belonged to the Society of Friends and were

born in Wales prior to 1682. In that year Dr. Edward Jones settled with his family in Merion Township, County of Philadelphia, Pennsylvania.

Young John Jones, after early education from his parents and at a private school in New York, began the study of medicine in Philadelphia. It was under the direction of Dr. Thomas Cadwallader (1708–1779)* and his own father, who practiced medicine in Philadelphia for a time before he removed to Long Island.

STUDIES IN EUROPE

Jones keenly felt the lack in this country of free institutions for the care of the sick, wherein the practice of medicine could be learned by practical methods at first hand. He therefore did what most of the better educated physicians of his day did. He went to Europe for further study. He was first at London where he attended the lectures of John Hunter (1728–1793) (who it will be remembered was himself a military surgeon, having served as Deputy Surgeon General of the British Army in 1786), James McKenzie (1680–1761) and other shining lights of the day. He likewise followed the practical clinical work in several hospitals, particularly Saint Bartholomew's, at which Percival Pott (1714–1788) (of Pott's fracture and Pott's disease) was senior surgeon, then at the peak of his reputation.

After some months in London, Jones went to France for further study. His thesis, *Observations on Wounds*, was published in New York in 1765, it is said, but no copy is known. He obtained his degree of Doctor of Medicine at the University of Rheims in May, 1751. Both before and after that date he studied at Paris where he remained until April, 1752. In Paris he

* Thomas Cadwallader was John Jones's first cousin, the former's aunt being the latter's mother. Cadwallader was educated in Europe and became one of the physicians of the Philadelphia Hospital at its establishment in 1752. He was a pioneer in inoculation. He is best known for his *Essay on the West-India Dry-Gripes*, printed by Benjamin Franklin in 1745. It is the first account of lead poisoning from rum distilled through lead pipes. He was twenty years older than Dr. Jones.

followed the anatomical lectures of Jean-Louis Petit (1674–1750) as well as the clinical work at the Hôtel Dieu, where Claude-Nicolas Le Cat (1700–1768) and Henri-François Le Dran (1685–1770) were then the leading surgeons. Afterward he spent some months at the University of Leyden where the great Boerhaave was his preceptor. Later he was for some months in Edinburgh where he completed his medical education, studying under the elder Alexander Monro (1697–1767). Certainly one must reckon John Jones an unusually well educated man.

Commenting on Jones's European training, his pupil and biographer, Dr. James Mease (1771–1846), who himself served in the War of 1812 as Hospital Surgeon, U. S. Army, said:

"Under masters like these, and enjoying such opportunities, he could not fail, with the assiduity which he employed, of acquiring all the knowledge of that time to be obtained: in consequence of the zeal which he showed in the acquisition of every species of useful knowledge, he attracted the notice of the above celebrated surgeons, which was of essential benefit to him, in the prosecution of his studies; to Mr. Pott, in particular, he considered himself under peculiar obligations, for the marks of friendship he experienced from him, and which could not fail to inspire him with a love of his profession.

"During the prosecution of his studies in Europe, Dr. Jones was industrious in collecting all the useful information in his power, especially upon those branches of science more immediately connected with his profession. To anatomy, as the handmaid of surgery, and the basis of medical science, he paid the greatest attention, but he did not suffer this to engross the whole of his time: convinced of the intimate connection between the different branches of the profession, he considered the separation of them, not only as contrary to nature, but as highly detrimental to the progress of the science; and therefore believed, that a knowledge of one part was indispensably

requisite to a right understanding of the other."

PRACTICE OF SURGERY IN NEW YORK

With the medical learning and skill he had gained in Europe, Jones began practice of surgery in New York. He early acquired a reputation as a skillful and speedy operator. This last was of paramount importance in those days of no anesthetics. Dr. Jones was the first surgeon to perform the operation of lithotomy in New York, and on this success much of his fame was built. He required but three minutes for the operation. "This operation," says Mease, "had likewise been frequently attempted in the other states, but the want of success attending it, was generally so great, as to prevent it from being performed in future: the fortunate manner, however, in which those cases under his care succeeded, fully proved, that it was no longer the dangerous operation many had been made to apprehend, and which induced them rather to submit to a miserable life, than suffer the risque of falling a sacrifice to the means instituted for their relief."

SERVICE IN THE FRENCH AND INDIAN WAR

In 1758, there burst forth in the British Colonies of North America that phase of the Seven Years' War which is usually known as the French and Indian War. Early in the conflict a report reached New York City of the intended attack of the enemy upon the frontiers of the Colony. Dr. Jones at once offered his services as a volunteer surgeon to the troops raised for the Colony's defense. He served in this capacity until the end of the war.

His reputation was so well established that he was well known to the French commander, General d'Escaux, who was dangerously wounded during the severe repulse suffered by the French in the engagement at Lake George, with the British and Colonial troops under Sir William Johnson. General d'Escaux, a prisoner of war, asked that he be placed under the care of Surgeon Jones while being treated for his wound. This was done and Jones attended him for a considerable time. At the close of the French and Indian War, Jones returned to private practice in New York City, his reputation being much increased by his military service.

FIRST PROFESSOR OF SURGERY AT KING'S COLLEGE

In 1767, the medical school of King's College, New York, was created. It was America's second pioneer school. This institution is now Columbia University. Jones was appointed the first occupant of the chair of surgery, thus becoming the second full professor of surgery in the country.* He was associated in the launching of the new medical school with such men as Samuel Bard (1742–1821) whose *A Discourse upon the Duties of a Physician* (1769), is the first American publication on medical ethics, and Peter Middleton (–1781) whose *A Medical Discourse or an Historical Inquiry into the Ancient and Present State of Medicine* is America's first book on medical history (1769).

As a professor, Jones displayed the ability of transmitting to his students the results of his own experience as well as of his European studies. He delivered his introductory address on November 9, 1767. "He endeavoured," we read, "to instill into the minds of his pupils the same just principles that guided his conduct. . . . Viewing the science in an enlarged and honourable light, as comprehending the most extensive view of our nature, and as tending to the alleviation and abridgement of human misery, he taught his pupils to despise the servile conduct of those who consider the profession as worthy of cultivation, only in proportion to the emoluments which it yields; and to rely upon the solidity of their own endowments, as

* The first professor of surgery in what is now the United States was Dr. William Shippen (1736–1808), sometime Director General of Military Hospitals of the Continental Army. He was appointed Professor of Surgery at the College of Philadelphia (now University of Pennsylvania) in 1766.

the best security of general esteem, and for the acquisition of business." (Mease) Translated into twentieth century expres-

JOHN JONES M.D.

FIG. 1. Portrait of Surgeon John Jones of the Continental Army. (From Mease's biography of Dr. Jones.)

sion, we gather that Dr. Jones was a man of correct conduct and teaching.

INTRODUCTORY LECTURE, KING'S COLLEGE, 1769

Mr. W. B. McDaniel, 2d, Librarian of the College of Physicians of Philadelphia, has published the full text of Jones's first lecture delivered at King's College, 1769, together with interesting notes. McDaniel calls attention to the rarity of manuscripts of the didactic lectures which were the customary method of teaching medicine in Jones's day, and for a long time thereafter. It is, therefore, most interesting and fortunate that this valuable manuscript has been preserved in the Library of the College of Physicians of Philadelphia. The volume containing it has also the nine later lectures of John Jones and his notes on the lectures of William Hewson (1739–1774). The manuscript volume was presented to the Library in 1902 by Dr. George W. Norris, to whom it had been presented, as a

penciled note states "by Mrs. Randolph." On the fly-leaf, in ink, in the manner of a title-page, is written: "Lectures on Surgery by the late John Jones, M. D., presented to me by Mrs. Clark." McDaniel thinks this is in the handwriting of Dr. James Mease, whose signature appears in the upper right-hand corner.

"The Introductory Lecture (of Jones) may be regarded as a companion piece to Peter Middleton's *Medical Discourse or an Historical Inquiry into the Present State of Medicine*, delivered at the opening of the medical school of King's College and printed in 1769; and to Samuel Bard's *First Commencement Address*, printed in the same year." (McDaniel)

Parts of this Introductory Lecture are almost the same as passages used later by Jones in his book. There are also other bits of advice to future practitioners which make good reading even today. For instance:

"This ancient branch of medicine called Surgery, according to the strict grammatical meaning of the word, signified manual operation, but the science & art of surgery, tho' more clear & certain in its objects, than that of Physic, is equally various, extensive & difficult of attainment. And I have ever been of opinion that young Physicians might lay the foundation for medical knowledge, by an attentive observation of those disorders, which surgery presented to their view. For most of those diseases, which are incident to the viscera & internal parts, are seen upon the external surface of our bodies, & are obvious to the senses, and the curative indications, as well as methodus medendi, in both have a very close analogy."

It had not been long since the surgeons were separated from the barbers, so that we are not surprised to read this conclusion to Jones's Introductory Lecture:

"To conclude, Gentlemen, if the science of surgery, then, requires genius, knowledge, & indefatigable application, to render its Professors truly respectable, what must we think of the insolence & malevolence of

those who represent it as a low mechanical art, which may be taught a butcher's boy in a fortnight—yet such false & absurd representations have been made of it, by some who have enjoy'd no small share of medical reputation in this country, & what is equally reproachful, there have been men who stil'd themselves gentlemen, ignorant & weak enough to credit such absurdities."

Jones continued his annual lectures until interrupted by the American Revolution.

FOUNDER OF THE NEW YORK HOSPITAL

In 1770, Dr. John Jones, with his fellow professor at King's College, Dr. Samuel Bard (1742–1821), and others petitioned King George III, through the Governor, John, Earl of Dunmore, for a charter for a hospital. The city of New York was then a town of about 20,000 inhabitants, scarcely extending as far north as St. Paul's Church. Jones and Bard had urged the hospital at the commencement of King's College in Trinity Church on May 16, 1769, and at their instance the Governor of the Province of New York, Sir Henry Moore, opened a subscription. Nearly a thousand pounds were raised in this way.

At the first meeting of the Governors of the New York Hospital, held in Bolton's Tavern on Wednesday, July 24, 1771, the charter was produced and read and the new institution formally came into being. On October 25, 1774, they appointed as their "Physicians in Ordinary" Doctors Peter Middleton, John Jones, Samuel Bard, and Malachi Treat.

The Minutes of the Governors of March 27, 1772, empowered, under the seal of the corporation, Dr. John Jones, then "intending for Europe," to make collections of money, and to buy medicines and apparatus abroad. The Secretary was directed to advertise in the public papers for plans. On July 2, 1773, the Governors ordered that the plan brought from England and laid before the Board by Dr. Jones, be carried into execution. They undertook but one story at first, for their finances were low. Jones had carried out the wishes of the Governors while in London and, despite his impaired health, had been able to collect moneys for the hospital.

Jones gave sound advice as to the construction of the new hospital. He had seen the terrible conditions at the Hôtel Dieu in Paris, where he found beds placed in triple rows, with four to six patients in each bed, and where more than once on the morning rounds he saw the dead lying with the living. The mortality, he said, was one-fifth of the whole number of patients received. He wrote to the Governors, "It is to be hoped that the hospital lately built in the City of New York will have fewer objections to its plan than any hospital hitherto constructed. The principal wards, which are to contain not more than eight beds, are thirty-six feet in length, twenty-four feet wide and eighteen high. They are all well ventilated, not only from the opposite disposition of the windows, but proper openings in the side walls, and the doors open into a long passage or gallery, thoroughly ventilated from north to south.

"If great and populous cities have been justly styled the graves of the human species, the large and crowded hospitals generally built in them may with equal truth be deemed the lazarettos or pest houses of the most unfortunate persons, who, from ill directed motives of compassion, are carried into these charities." (From the Appendix to Jones's *Plain Concise Remarks*, etc.)

One of the medical tragedies of the period is the destruction by fire of the New York Hospital. "On Tuesday last," says the *New York Gazette and Weekly Mercury* of March 6, 1775, "between twelve and one o'clock, the new hospital at Ranelagh, a large pile of buildings lately erected and nearly finished, was discovered to be on fire, the workmen being all gone to dinner, and the whole wooden part of the building was, in about an hour, reduced to ashes." The building was rebuilt but it was January 3, 1791, a few months before Jones's death, before the wards were actually open for the reception of patients.

SECOND VISIT TO BRITAIN

Jones, we are sad to read, was an asthmatic. He had "long struggled to over-

ineffectual towards even his relief." There-fore, he decided on a voyage to Europe for his health, and sailed for London.

PLAIN CONCISE

PRACTICAL REMARKS

ON THE TREATMENT OF

WOUNDS AND FRACTURES;

TO WHICH IS ADDED, A SHORT

A P P E N D I X

ON

CAMP AND MILITARY HOSPITALS;

PRINCIPALLY

Defigned for the Ufe of young MILITARY SURGEONS, in NORTH-AMERICA.

By JOHN JONES, M. D.
Profeffor of Surgery in King's College, New York.

N E W - Y O R K:
Printed by JOHN HOLT, in Water-Street, near the Coffee-Houfe.

M,DCC,LXXV.
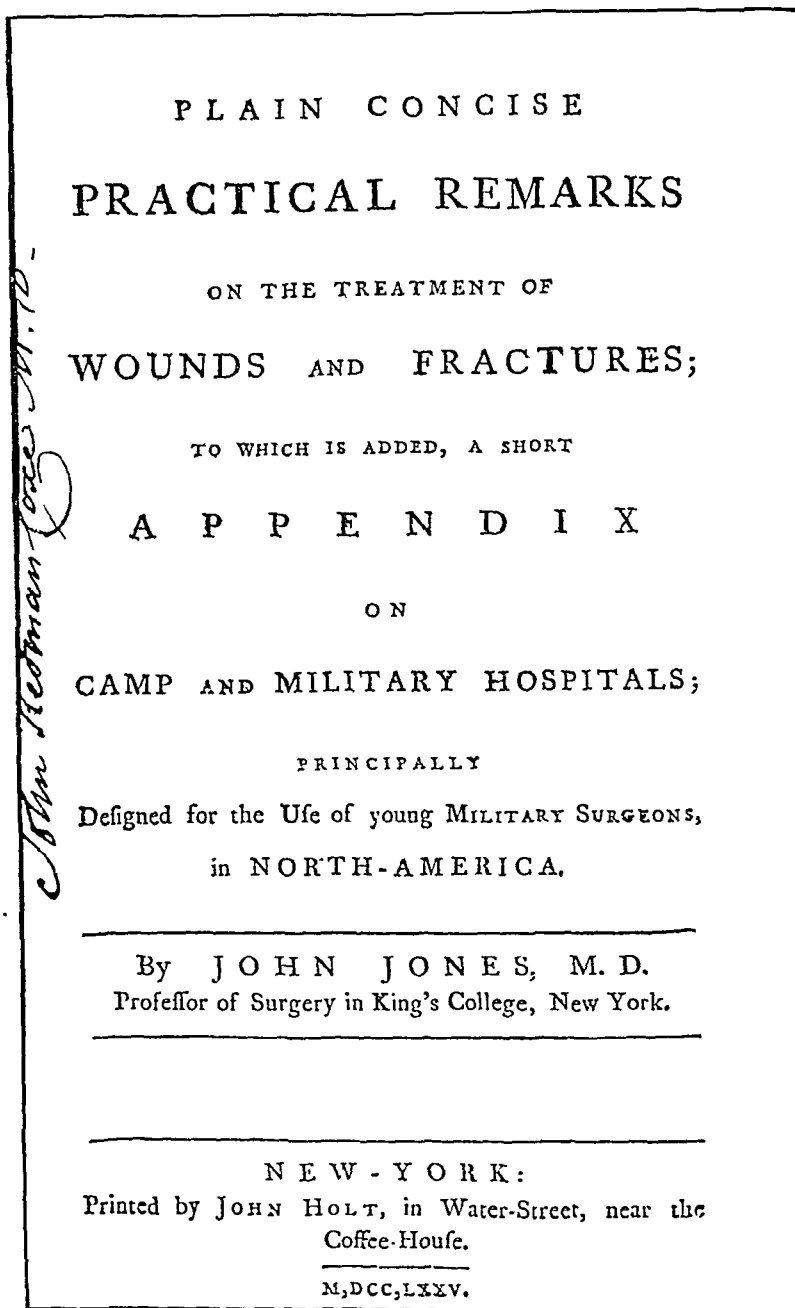
FIG. 2. Title page of the first edition of Surgeon Jones's Monograph, 1775. This is the first American medical book.

come that painful disease," but "the exertions of both his own skill, and of the rest of his medical brethren, in most parts of the continent" had unfortunately "proved

One may not quite follow the therapeutic advice on which this was based. In London, wrote Mease, "in the thick smoke, and an atmosphere impregnated with every species

of animal putrefaction and effluvia, where so many asthmatics have found such re-

remainder of his life was attributed to the health-giving air of London!

PLAIN CONCISE

PRACTICAL REMARKS,

ON THE TREATMENT OF

WOUNDS AND FRACTURES;

To which is Added, An APPENDIX,

ON

CAMP AND MILITARY HOSPITALS;

PRINCIPALLY

Defigned, for the Ufe of young Military and Naval Surgeons, in NORTH-AMERICA.

By JOHN JONES, M. D.
Profeffor of Surgery, in King's College, New York.

PHILADELPHIA:
Printed, and Sold, by ROBERT BELL, in Third Street,

MDCCLXXVI.
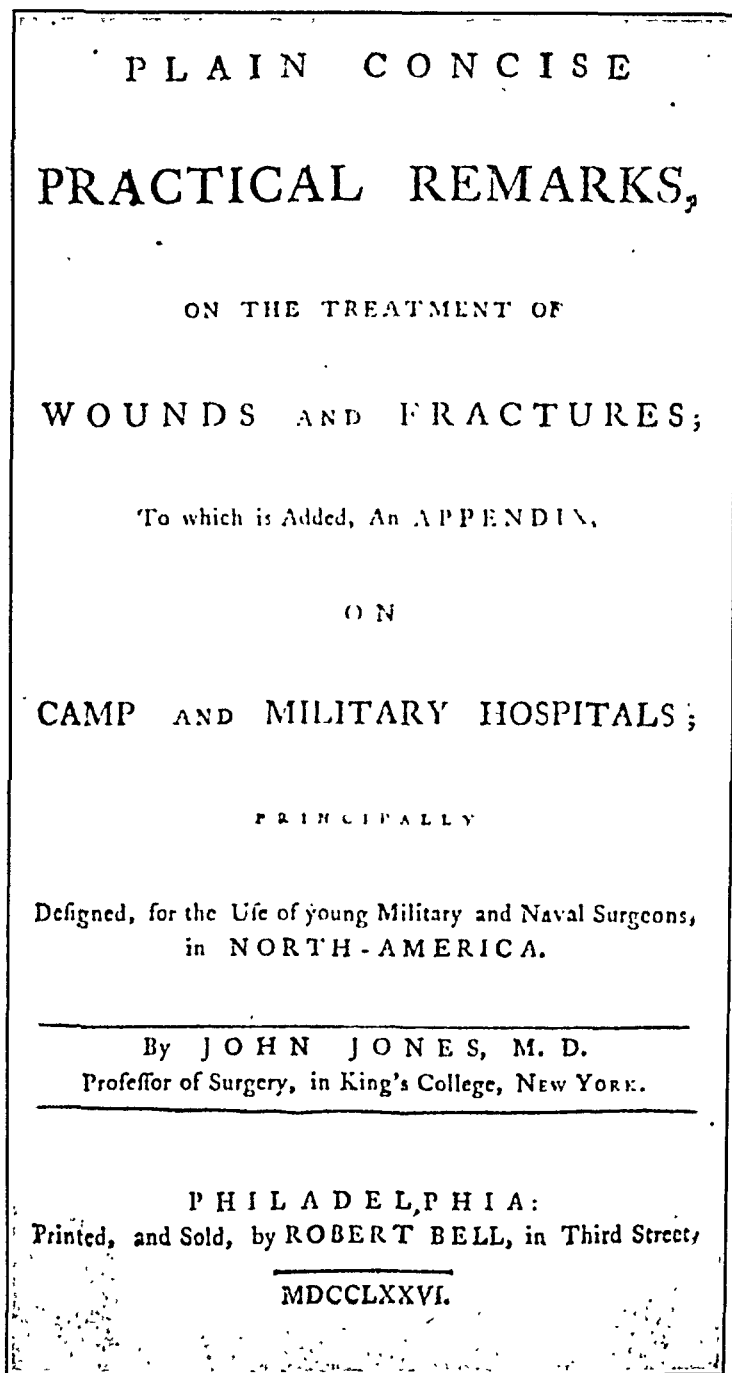
Fig. 3. Title page of the second edition of Surgeon Jones's Monograph, 1776. Published one year after the first edition, the book is now designed for the use of naval as well as military surgeons.

markable benefit, he also experienced a considerable alleviation of his complaint." Indeed his improved state of health for the

In London, he had once more the privilege of seeing some of his old mentors, particularly Percival Pott. Jones, we learn

with pleasure, "unlike many who suppose all knowledge to become stationary at the time of their leaving college, was still willing to be taught by those who had formerly been his instructors." Full well did he realize that they "from the greater opportunities they enjoyed, would be able to afford him much information."

Jones visited John Hunter again and met with a cordial reception. From Pott he received a complete copy of his celebrated lectures. Pott also made a practice of recommending Jones to those who wrote him for his opinion, and to travelers destined for New York. This had much to do with the spread of Jones's fame.

AMERICA'S FIRST SURGICAL BOOK

In 1775, he wrote his *Plain Remarks upon Wounds and Fractures*, which he inscribed to Dr. Thomas Cadwallader, his kinsman and preceptor. The book was a welcome one indeed. It contains much material based on Pott's teachings, and that of other of Jones's European professors. Jones drew somewhat, also, on the work of John Ranby (1703–1773), Serjeant-Surgeon to the King. It will be recalled that in 1745 Ranby had been responsible for the act that formally separated the surgeons from the barbers as "Masters, Governors, and Commonality of the Art and Science of Surgeons of London," which later became the Royal College of Surgeons of England.

For all this, Jones's book contained original observations. "Few presents could have been more acceptable to his country, or more opportunely made; for, in the then situation of American affairs, many persons were chosen to act as surgeons, who from their lamentable ignorance of the principles of their profession were but ill qualified for the office." Billings notes Jones's having called attention to *hernia cerebri* following trephining.

It was immediately hailed as a valuable and timely work, a veritable *vade mecum.* The War for Independence was beginning and among the many needs of the new army was a great demand for surgeons.

There were comparatively few possessors of medical degrees in the Colonies at that time, though many other persons practiced medicine without degrees. Unfortunately for the medical service, many of the best qualified physicians became officers of the line instead of performing medical duties. For instance, General Warren, who fell at Bunker Hill, General St. Clair, who commanded Pennsylvania troops, General Hugh Mercer, who fell at Princeton, Colonel Theodoric Bland of Virginia, and many more, were physicians.

The value of Jones's monograph was enhanced by binding with it his translations of Gerard L. B. van Swieten's (1700–1772) *Diseases Incident to Armies.*

A second edition appeared in the following year: "Plain, Concise, Practical, Remarks on the Treatment of Wounds and Fractures; To which is Added, an Appendix on Camp and Military Hospitals. Principally designed for the use of young Military and Naval Surgeons in North America. Philadelphia, R. Bell, 1776," 114 pages. Four years after his death a third edition was published by Jones's friend Dr. James Mease (1771–1846). It is entitled:

"The Surgical Works of the late John Jones, M.D. Formerly Professor of Surgery in the College of New York, Fellow of the American Philosophical Society, President of the Humane Society, and Vice-President of the College of Physicians of Philadelphia, Physician of the Pennsylvania Hospital, and Philadelphia Dispensary. The third edition. To which are added a short account of the life of the author with occasional notes and observations. By James Mease, M.D., Resident Physician of the Port of Philadelphia. Printed by Wrigley & Berriman, Philadelphia, 1795." A copy of this work, presented by the author, is in the collection of the Library Company of Philadelphia.

Jones's attitude toward his duty as a military surgeon is reflected in his dedication of his famous book to his preceptor and cousin, Dr. Thomas Cadwallader: "If I cannot cure the fatal diseases of my

unfortunate country, I can at least pour a little balm into her bleeding wounds." a surgeon, and on the duties of that profession. He says:

THE

SURGICAL WORKS

OF THE LATE

.JOHN JONES, M. D.

FORMERLY PROFESSOR OF SURGERY IN THE COLLEGE
OF NEW-YORK:
FELLOW OF THE AMERICAN PHILOSOPHICAL SOCIETY:
PRESIDENT OF THE HUMANE SOCIETY, AND VICE-PRESIDENT
OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA:
PHYSICIAN TO THE PENNSYLVANIA HOSPITAL,
AND PHILADELPHIA DISPENSARY.

The Third Edition.

TO WHICH ARE ADDED,

A Short Account of the Life of the Author,

WITH

OCCASIONAL NOTES AND OBSERVATIONS.

By JAMES MEASE, M. D.

RESIDENT PHYSICIAN OF THE PORT OF PHILADELPHIA.

PHILADELPHIA:

PRINTED BY WRIGLEY AND BERRIMAN,
N°. 149, Chefnut Street.

1795.

FIG. 4. Title page of the third edition of Surgeon Jones's Monograph, 1795. This final edition edited by Jones's pupil, Dr. James Mease, appeared four years after Jones's death.

In the introduction to his book, Jones gives the student and others some sound advice as to the proper manner of becoming "It will appear very evident how necessary it is for the student in Surgery to make himself thoroughly acquainted with

most of those branches of medicine, which are requisite to form an accomplished physician.

"Besides a competent acquaintance with the learned languages, which are to lay the foundation of every other acquisition; he must possess an accurate knowledge of the structure of the human body, acquired not only by attending anatomical lectures, but by frequent dissections of bodies with his own hands. This practice cannot be too warmly recommended to the students in Surgery: It is from this source, and a knowledge in hydraulics, they must derive any adequate notions of the animal œcon-omy of Physiology. Chymistry and Materia *Medica are very necessary to a right under-*standing of pharmacy or composition. To these should be added some progress in the mathematics and mechanics, which I will venture to assert, may be applied with much more utility and safety to the science of Surgery, than Physic. But there must be a happiness, as well as art, to complete the character of the great Surgeon.

"He ought to have firm steady hands, and be able to use both alike; a strong clear sight, and above all, a mind calm and intrepid, yet humane and compassionate, avoiding every appearance of terror and cruelty to his patients, amidst the most severe operations."

This sage council is as good today as when Jones gave it before the day of America's independence. He constantly urged thoroughness in study and practice. A superficial student will "not be likely to reap much benefit from scampering round the wards of an hospital, and reading a general system of Surgery." He says else-where in his text: "As to those gentlemen who will neither read nor reason, but prac-tice as a venture, and sport with the lives and limbs of their fellow-creatures, I can only, with Dr. Huxham, advise them seri-ously to peruse the fifth Commandment, which is 'Thou shalt not kill.'" On the other hand "knowledge ripened by experi-ence" is a "proper requisite of a good surgeon." To practitioners in the new

country, where opportunity is not within the reach of every student, he "earnestly recommends a diligent, attentive and re-peated perusal of the best English writers."

Though it is impracticable to give lengthy extracts from the text of Jones's monograph, the titles of the chapters give an insight:

I. Of Wounds in General
II. Of Inflammation
III. Of the Division of Wounds
IV. Of Penetrating Wounds of the Tho-rax and Abdomen
V. On Simple Fractures of the Limbs
V. On Compound Fractures [There are two chapters numbered v.]
VI. On Amputation
VII. Of Blows on the Head
VIII. On Injuries Arising from Concus-sion or Commotion
IX. On Injuries Arising from a Fracture of the Skull
X. Of Gunshot Wounds

To this is added the famous appendix, the first American text on hygiene. It is en-titled: "An Appendix Containing Some Short Hints on the Structure and Œconomy of Hospitals; Particularly Applied to Mili-tary Ones, with the General Means of Pre-serving Health in an Army." The appendix is twelve pages in length.

MEMBER OF THE NEW YORK SENATE

When the war that was to result in American independence began, Jones threw his considerable influence toward the party that insisted on the rights of the Colonists. When New York was occupied by the British Army, Jones escaped and shortly afterward became a member of the New York Senate. He served in this capacity as long as he believed that he was of use and then resigned so as to enter the military service.

MILITARY SERVICE IN THE AMERICAN REVOLUTION

Following his service as a member of the New York Senate, Dr. Jones was appointed Surgeon's Mate of the' Tenth Massachu-

setts Regiment of the Continental Line. A Surgeon's Mate in those days was what was later known as an Assistant Surgeon. On September 24, 1777, he was promoted Surgeon in the same regiment.

When Philadelphia was evacuated by the British troops in the summer of 1778, Surgeon Jones was sent there in an official capacity. This was much to his liking, not only on account of the importance of being in the metropolis of the country but because the climate seemed to improve his asthma. Whether this were on account of foul atmosphere similar to that of London in which he had improved, the record does not say.

Surgeon Jones continued in the military service until May 14, 1781, (Heitman) when he resigned and began the practice of medicine in Philadelphia, which was to remain his home for the rest of his life. His frail state of health prevented more active field service, but he is credited with an important share in organizing the medical department of the Continental Army. J. M. Toner. *Tr. Am. M. A.*, 29: 689, 1878.

JONES'S SUCCESS IN PHILADELPHIA

Jones met with the same success as a practitioner in Philadelphia that he had experienced in New York. "The same agreeable manners as a gentleman, which placed him so high in the esteem of his fellow-citizens at New York, could not fail of attracting those of his new place of residence," says Mease. He had been a member of the American Philosophical Society since his election on April 21, 1769. In January, 1786, he became a member of its council.

In 1780, he succeeded Dr. John Redman as one of the Physicians of the Pennsylvania Hospital. He held this position until his death. In the same year, he was elected a consulting physician of the Philadelphia Dispensary. When the Humane Society (for the resuscitation of drowned persons) was formed in 1780, Jones was elected its President. The other physicians who were members were Benjamin Rush, Benjamin

Duffield, Caspar Wistar, Samuel Powell Griffits, and John R. B. Rodgers (American Museum, 1787). All of these memberships attest Jones's high place in the community.

VICE-PRESIDENT OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

When the College of Physicians of Philadelphia was instituted in 1786, Dr. Jones was elected its first Vice-President, the Presidency being given to Dr. John Redman (1722–1808). By annual re-election, Jones held the office of vice-president to the end of his life, and might have succeeded to the presidency had he survived Redman. One of the best contemporary opinions of Jones is given in Redman's acknowledgment, August 2, 1791, of his re-election as President of the College.

Of his colleague, Jones, Redman said:

"I should not have been easy under the sense I had of my growing infirmities of body and mind for some time past, to have continued to accept the honor you have so repeatedly conferred upon me, but from the consideration that you always joined a colleague with me as Vice-President, whose eminence and reputation in our profession, and whose clearness of judgement, vigor of faculties, and easy manner of conveying his sentiments, together with his friendly disposition to aid me, fully obviated and prevented any ill effects, naturally to be expected from declining age, and rendered my situation more pleasant than otherwise it might have been. But though much and justly respected by us, and all connected with him in kindred, friendship, and business, he was mortal, and he has gone—no more to return, to aid by his talents, or gratify us by his presence at our meetings, or cheer us by his affability, agreeable converse, and polite manners. And, therefore (though somewhat late and unseasonable), I must indulge myself in sympathizing with you and regretting the real loss which the republic of medicine in general, and our collegiate society in particular, have sustained thereby. Much did I expect, from his being several years

younger than myself, and so well and justly esteemed by you, that he would be my next successor; and from a settled resolution soon to request my dismission (if not otherwise removed), I sometimes flattered myself with having the pleasure to see him raised to your presidential chair—to which I should most heartily have concurred—as well on account of his own merit and qualifications, as because it would have been highly gratifying to me to be a living witness of our College being headed by one whose eminence in more than one of the material branches of medical science, and reputation among our citizens in general was still very flourishing, and whose connection with and estimation in which he was held by the higher orders and rank of them, was so conspicuous and intimate, as might contribute to the greater external dignity of the institution, and render its influence more powerful and effectual on any particular occasion of public utility, wherein it might be thought requisite or be called upon to exert it." (*Transactions College Physicians Philadelphia*, 1887, 49–50.)

Jones contributed one of his most interesting papers, "A Case of Anthrax," to the first part of the first volume of the *Transactions of the College of Physicians*. (1793.) It was read posthumously.

BENJAMIN FRANKLIN'S PHYSICIAN

It is not surprising that so prominent a man as Jones has the greatest of Pennsylvanians as his patient. Benjamin Franklin was his intimate, personal friend and was under his medical care through the last years of his life. In his will, dated July 17, 1788, Franklin left Jones a bequest. Jones's friend, Dr. James Mease (1771–1846) who published memoirs of Jones's life, preserved the following brief account of Franklin's last illness. It was written down by Jones at the time and later published by him as "A Short Account of Dr. Franklin's last Illness," which appeared both in the *Pennsylvania Gazette* and *Freeman's Journal*, both of Philadelphia, for April 21, 1790. The following are passages:

"The stone, with which he had been afflicted for several years, had for the last twelve months of his life, confined him chiefly to his bed; and during the extremely painful paroxysms, he was obliged to take large doses of laudanum to mitigate his tortures; still, in the intervals of pain, he not only amused himself by reading and conversing cheerfully with his family and a few friends who visited him, but was often employed in doing business of a public, as well as of a private nature, with various persons who waited upon him for that purpose; and in every instance, displayed not only the readiness and disposition to do good, which were the distinguishing characteristics of his life, but the fullest, and clearest possession of his uncommon abilities. He also not unfrequently indulged in those jeux d'esprit, and entertaining anecdotes, which were the delight of all who heard them.

"About sixteen days before his death he was seized with a feverish disposition, without any particular symptoms attending it till the third or fourth day, when he complained of a pain in his left breast, which increased till it became extremely acute, attended by a cough and laborious breathing. During this state, when the severity of his pains drew forth a groan of complaint, he would observe, that he was afraid he did not bear them as he ought; acknowledging his grateful sense of the many blessings he had received from the Supreme Being, who had raised him from small and low beginnings, to such high rank and consideration among men: and made no doubt but that his present afflictions were kindly intended to wean him from a world in which he was no longer fit to act the part assigned him. In this frame of body and mind, he continued until five days before his death, when the pain and difficulty of breathing entirely left him, and his family were flattering themselves with the hopes of his recovery; but an imposthume [abscess] which had formed in his

lungs, suddenly burst, and discharged a quantity of matter, which he continued to throw up while he had power, but as that failed, the organs of respiration became gradually oppressed; a calm lethargic state succeeded; and on the 17th instant, [April, 1790] about eleven o'clock at night, he quietly expired, closing a long and useful life of eighty-four years and three months.

"It may not be amiss to add to the above account, that Dr. Franklin, in the year 1735, had a severe pleurisy, which terminated in an abscess of his lungs; and he was then almost suffocated by the quantity and suddenness of the discharge. A second attack, of a similar nature, happened some years after, from which he soon recovered; and did not appear to suffer any inconvenience in his respiration from these diseases."

Since Franklin remembered Jones in his will, we conclude that he had forgotten his own advice "He is a fool that makes his doctor his heir." (Poor Richard, 1733.)

DEATH OF JONES

President Washington and his family were patients of Dr. Jones. Indeed it was while on a professional visit to the Father of his Country, that Jones was stricken with his own last illness. He had been to see General Washington on the evening of June 17, 1791. It was a cool evening after a sultry day. "Dr. Jones was dressed in a light manner, suitable to the weather, when he set out; but it was not sufficiently warm for the remarkable and sudden alteration in the temperature of the air that succeeded." Feeling indisposed on his return home, he awoke next morning "with considerable fever, attended by diarrhœa, and great prostration of strength." (Mease.) There was a return of his old asthma, and the "conversation of his numerous friends who had visited him" added to the depletion of his strength. He became weaker and weaker and finally passed away in his sleep on June 23, 1791, being sixty-three years of age.

PERSONAL APPEARANCE

Jones was of about middle size, his "habit being thin, owing to his constant affliction with asthma." He had a quick and penetrating eye and a "cheerful but sedate countenance." His gravity of appearance and dignity never failed to command respect.

PROFESSIONAL POSITION

Best known as a lithotomist, he was a general surgeon and stood at the head of his profession in the United States. His technic of lithotomy became generally accepted and had much to do with the reduction in mortality that had theretofore attended the operation. He was likewise highly respected as an obstetrician. He is said to have given the first systematic instruction in midwifery to medical students in this country. He opposed the then too general use of drugs, being "convinced that nature, or more properly speaking, the exertions of the system, were, in the greatest number of instances, sufficient for its own necessities." Though educated in the school of Boerhaave, he never gave himself to any particular "system." He was ever ready to accept any rational method of treatment which had the sanction of experience.

Dr. S. Weir Mitchell, in his Commemorative Address at the centennial of the College of Physicians of Philadelphia, thus sums up his opinion of Jones:

"John Jones was of the Society of Friends, and lies, since 1791, after their fashion, in a nameless grave under the maples of their Arch Street burial ground. He was a man tranquil of temper, easy and polite, fond of poetry and belles-lettres, a surgeon so expert in lithotomy that he frequently operated for stone in a minute and a half."

"He was," says Ruschenberger, "generally considered to be the foremost American surgeon of his day, noted for the prudence of the plan and the celerity of his operations, a quality very highly valued before the introduction of anaesthetics.

"Few persons possessed more of those engaging qualities which render a man estimable, both professionally and otherwise, than Dr. Jones. His conversation was most pleasing. His language flowed in an easy, spontaneous manner, and was animated by a vein of sprightly but always unoffending wit, which delighted while it secured attention. He was a *belles-lettres* scholar, was observant, and possessed a good memory; and was ever a most agreeable, entertaining, and instructive companion."

REFERENCES

1   JONES, JOHN. Plain Concise Practical Remarks on the Treatment of Wounds and Fractures, to Which is Added, a Short Appendix on Camp and Military Hospitals, Principally Designed for the Use of young Military Surgeons in North-America, 93 pages New York, 1775

2   JONES, JOHN. Plain Concise Practical Remarks, on the Treatment of Wounds and Fractures, To Which is Added, an Appendix on Camp and Military Hospitals, Principally Designed for the Use of young Military and Naval Surgeons in North-America 2nd ed 114 pages Philadelphia, 1776 [Note that while the former edition is designed for the use of young military surgeons, the latter is likewise for young naval surgeons]

3. MEASE, JAMES The Surgical Works of the late John Jones, M D , Formerly Professor of Surgery in the College of New York, Fellow of the American Philosophical Society, President of the Humane Society, and Vice-President of the College of Physicians of Philadelphia, Physician of the Pennsylvania Hospital, and Philadelphia Dispensary. Third Edition To which are added a Short Account of the Life of the Author, with Occasional Notes and Observations. 3rd ed. Philadelphia, 1795.

4. JONES JOHN. A Case of Anthrax. *Tr. Coll. Phys. Phila.*, 1: 1, 1793.

5   *Gaz. U. S.*, Philadelphia, June 25, 1791.

6. *Penn Gaz.*, Philadelphia, June 29, 1791.

7. Account of the Life and Character of the late John Jones, M.D., Formerly Professor of Surgery in King's (now Columbia) College, New York &c., *American Medical and Philosophical Register*. New York, 3: 325–337, 1814.

8. THATCHER, JAMES. *American Medical Biography*, Boston, 1828

9   BECK, JOHN B An Historical Sketch of the State of American Medicine Before the Revolution. (Annual Address before the Medical Society of the State of New York, February 1, 1842, Albany.)

10 BEEKMAN, JAMES WILLIAM. Centenary Address Delivered Before the Society of the New York Hospital, July 24, 1871.

11. BILLINGS, JOHN SHAW. A Century of American Medicine, 1876.

12 TONER, JOSEPH M. *Tr. A M. A.*, 29· 689, 1878.

13. RUSCHENBURGER, W. S. W. An Account of the Institution of the College of Physicians of Philadelphia. *Tr. Coll Phys. Phila.*, 3rd Series, 60. 33, 1887.

14 MITCHELL, S WEIR. Commemorative Address at the Centennial of the Institution of the College of Physicians of Philadelphia, *ibid*, 337.

15. HENRY, F. P. Standard History of the Medical Profession of Philadelphia, 1897.

16 SCHROEDER, WILLIAM, SR , *Bklyn. Med. J.*, 15: 473, 1900.

17. HEITMAN, F. B. Historical Register of Officers of the Continental Army, 1775–1783, 2nd ed , p. 324, 1914.

18 SMITH, STEPHEN. History of Surgery. Reference Handbook of the Medical Sciences, 4th ed., 8: 46.

19. BUCK, ALBERT H. Sketch of John Jones, *ibid*, 5. 705–706.

20. WEAVER, GEORGE H. Dr. John Jones, 1727–1791. *Bull. Soc. Med Hist. Chicago*, 2: 191–195, 1919.

21. PREBLE, EDWARD. Dictionary of American Biography, 10: 181–182.

22 LOBINGIER, ANDREW STEWART. Master Surgeons of America—John Jones. *Surg , Gynec. & Obst*, 51: 740–743, 1930

23 McDANIEL 2d , W. B. John Jones' Introductory Lecture to His Course in Surgery (1769), King's College, Printed from the Author's Manuscript *Transactions and Studies of the College of Physicians of Philadelphia*, December, 4th series, 8· 180–190, 1940.

IS THIS THE TRANSFUSION PICTURE IN YOUR HOSPITAL ?

With a mental picture of such a major procedure before him, small wonder that many a doctor orders transfusion only as a last resort. The cost is high. Danger of contamination is great. *"Maybe the patient can get by without it."*

A section of the stringing department

Equipment for testing and recording tensile strength

Swaging Atraumatic needles to special-purpose sutures

Chemical research laboratory
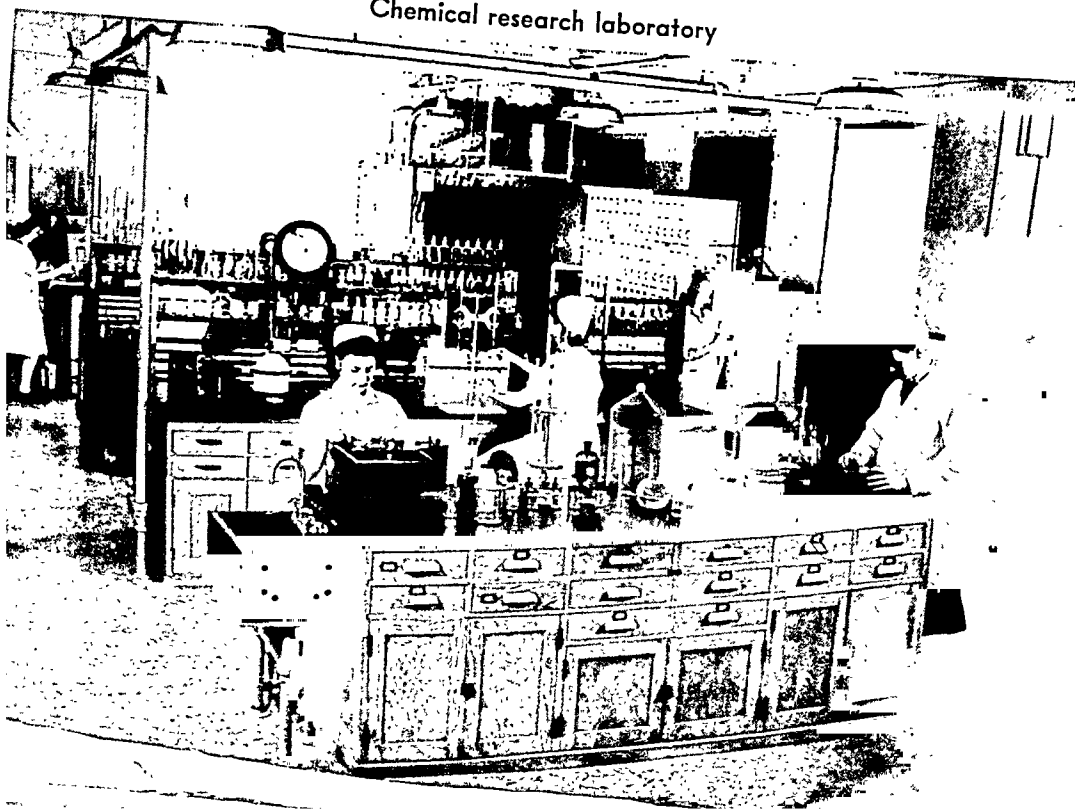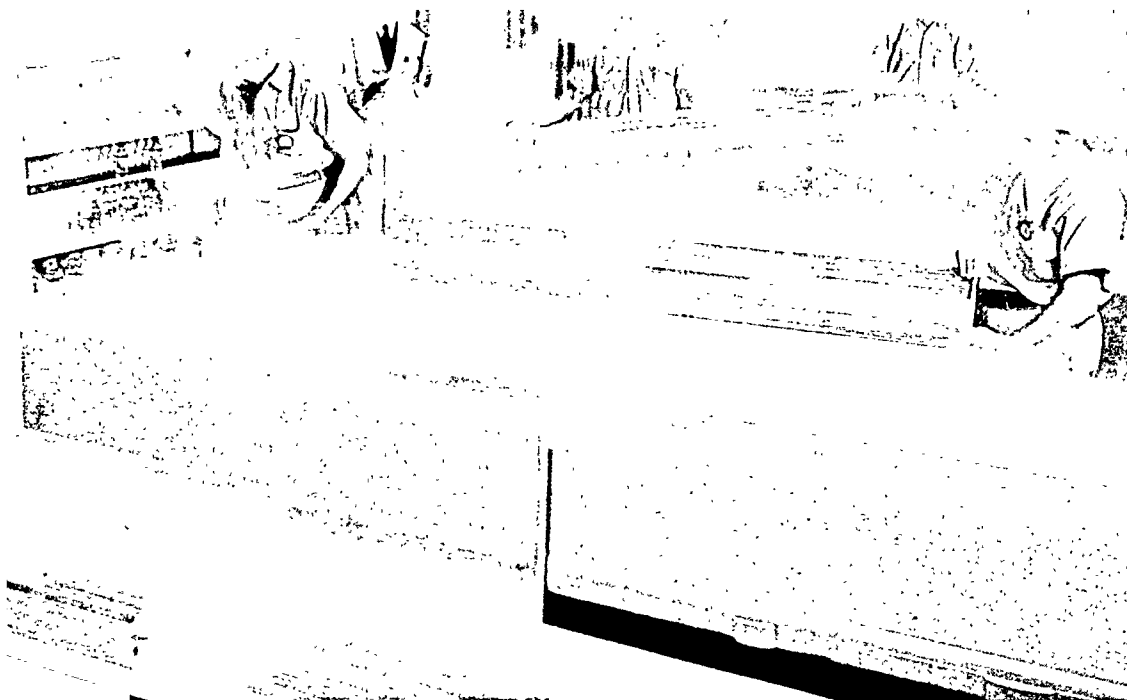
A step in the neutralizing process

Part of the winding department
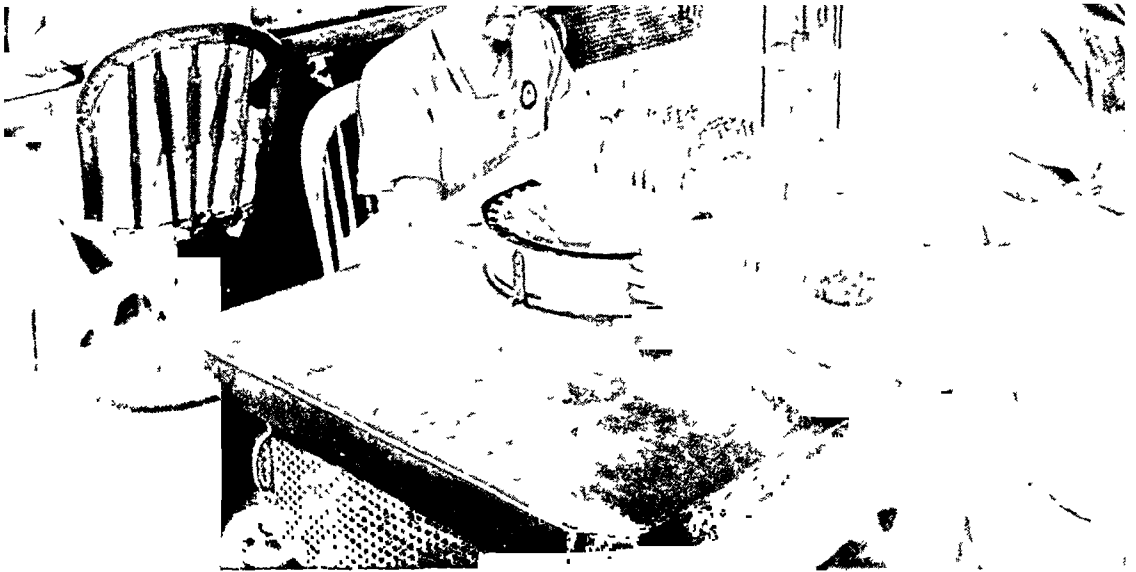
Two of our special machines for automatically adding storing fluid and sealing suture tubes

A battery of electric autoclaves for high-heat sterilization
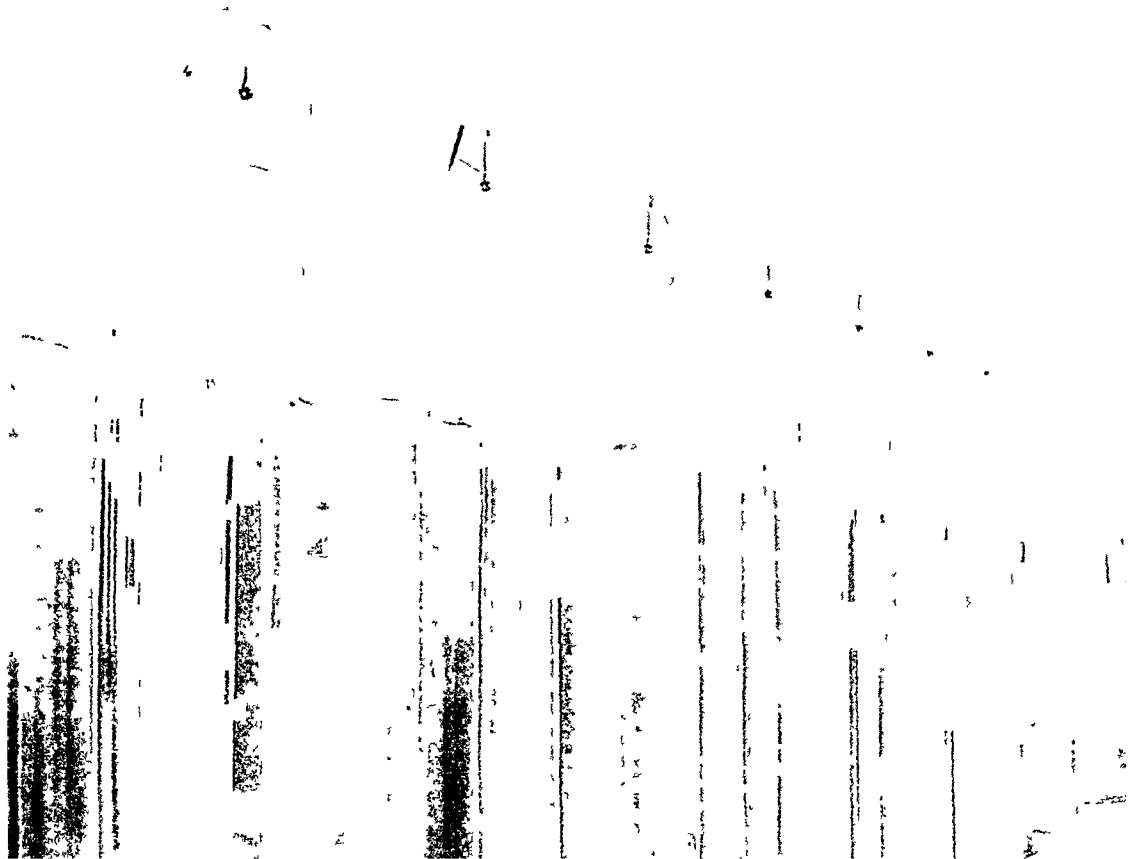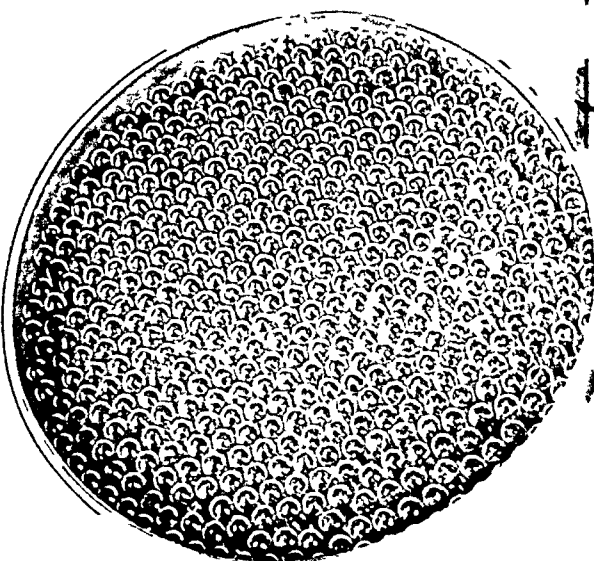
Glass cabinets protect in-process sutures
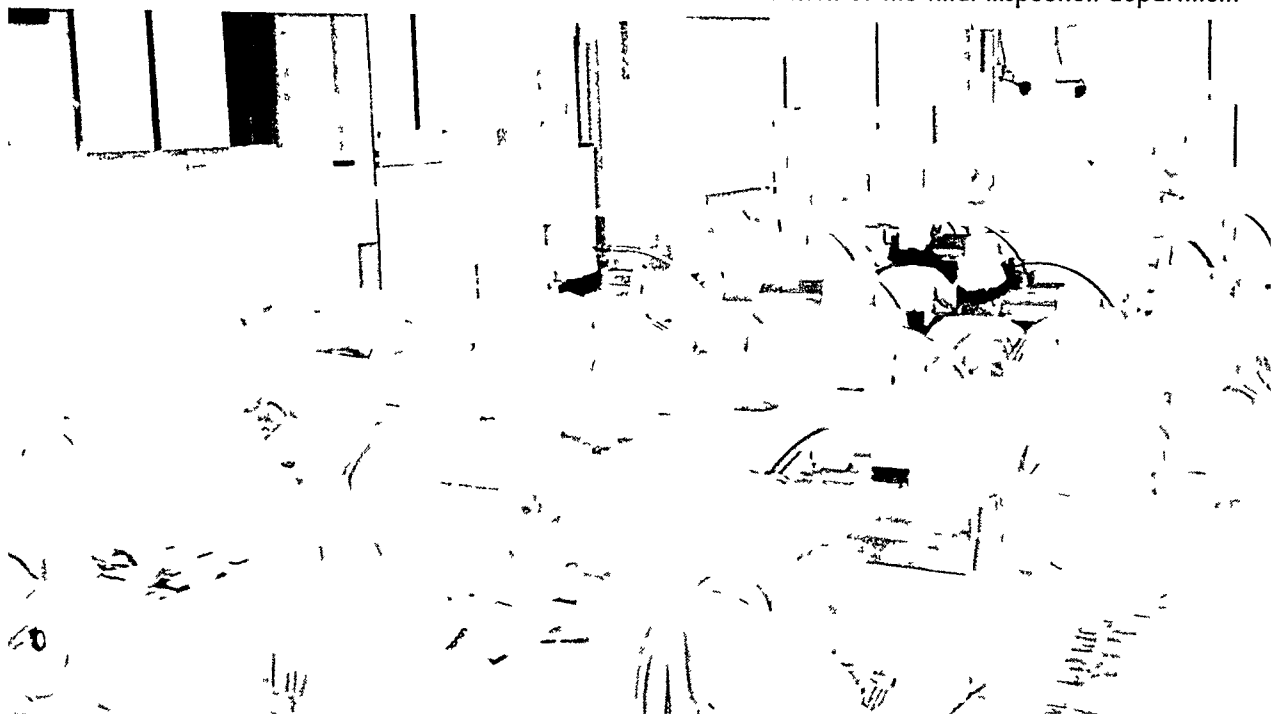
A division of the final inspection department
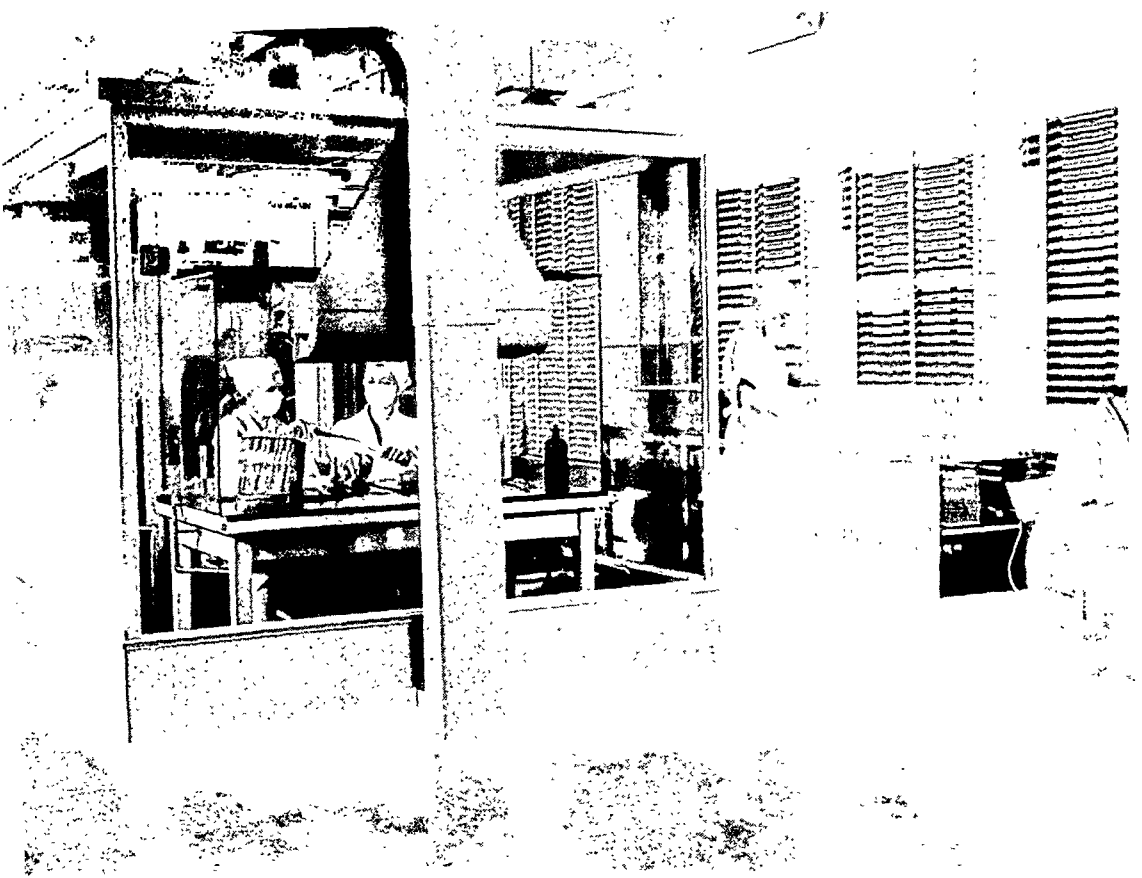
Dust-proof glass-enclosed chamber in the bacteriologic testing department

Expansion of our manufacturing facilities
has been paralleled by intensification of our research activities.
The rapidity of developments in all scientific fields
is making this work unusually productive.
Therefore, assurance can be given not only for adequate quantities
but for advances in processing
and still further refinements in the products themselves.

57 WILLOUGHBY STREET · BROOKLYN, N.Y., U.S.A.

IN THYROID SURGERY

Safer Patient Preparation

The record of ill prepared patients sent to thyroid surgery because preoperative medication had to be stopped due to iodine reaction, is not an inspiring one. Frequently such patients have proved to be as poor risks as if they went to surgery entirely unprepared.

Amend's Solution solves this problem. It combines high therapeutic efficacy with dependable safety. It is well tolerated, even by patients known to be sensitive to Lugol's solution or the iodides. In many instances in which other iodine preparations had to be withdrawn, Amend's Solution has permitted continuation of therapy and led to rapid disappearance of all signs of intoxication.

A stable, aqueous (1.21%) solution of resublimed iodine, largely in organic form, Amend's Solution contains no glycerin, or alcohol. It is available upon prescription through all pharmacies in 2-ounce bottles.

The American Journal of Surgery, Inc.

Contents · February, 1943

The American Journal of Surgery

Contents • February, 1943

General Information

THE AMERICAN JOURNAL OF SURGERY does not hold itself responsible for any statements made or opinions expressed by any contributor in any article published in its columns.

Articles are accepted for publication with the understanding that they are original contributions never previously published. All manuscripts are subject to editorial modification, and upon acceptance become the property of THE AMERICAN JOURNAL OF SURGERY, INC.

A reasonable number of illustrations are supplied free of cost; special arrangements must be made with the editor and publishers for excess illustrations and elaborate tables.

Reprints are furnished on order. Prices are quoted when articles are in page form.

Material published in THE AMERICAN JOURNAL OF SURGERY is copyrighted and may not be reproduced without permission of the publishers.

Change of address must reach us by the 15th of the month preceding month of issue.

PREPARATION OF MANUSCRIPTS

Text. Manuscripts are to be typewritten, on one side of the paper, with double spacing and good margins. The original should be sent to the editor and a carbon copy retained by the author.

Illustrations. Illustrations must be in the form of glossy prints or drawings in black ink (*never* in blue). On the back of each illustration the figure number, author's name and an indication of the top of the picture should be given. Legends for illustrations are to be typewritten in a single list, with numbers corresponding to those on the photographs and drawings. Please do not attach legends to the pictures themselves.

Bibliographies. Bibliographic references should be at the end of the manuscript, in alphabetical order, and not in footnotes. Each reference should include the following information in the order indicated: Name of author with initials; title of article; name of periodical; volume, page and year. The following may be used as a model:

PHANEUF, LOUIS E. Indications and technique. Am. J. Surg., 25: 446, 1937.

The author should always place his full address on his manuscript.

Address all correspondence to

The American Journal of Surgery, Inc.

49 WEST 45TH STREET · NEW YORK

← HIGH **GERMICIDAL ACTIVITY**

LOW TOXICITY TO TISSUE*

1:5250	1:117	1:1250**	1:1200	1:120	1:300	1:125**	1:2250
CEEPRYN		MERCURIAL # 1		PHENOL		MERCURIAL # 2	
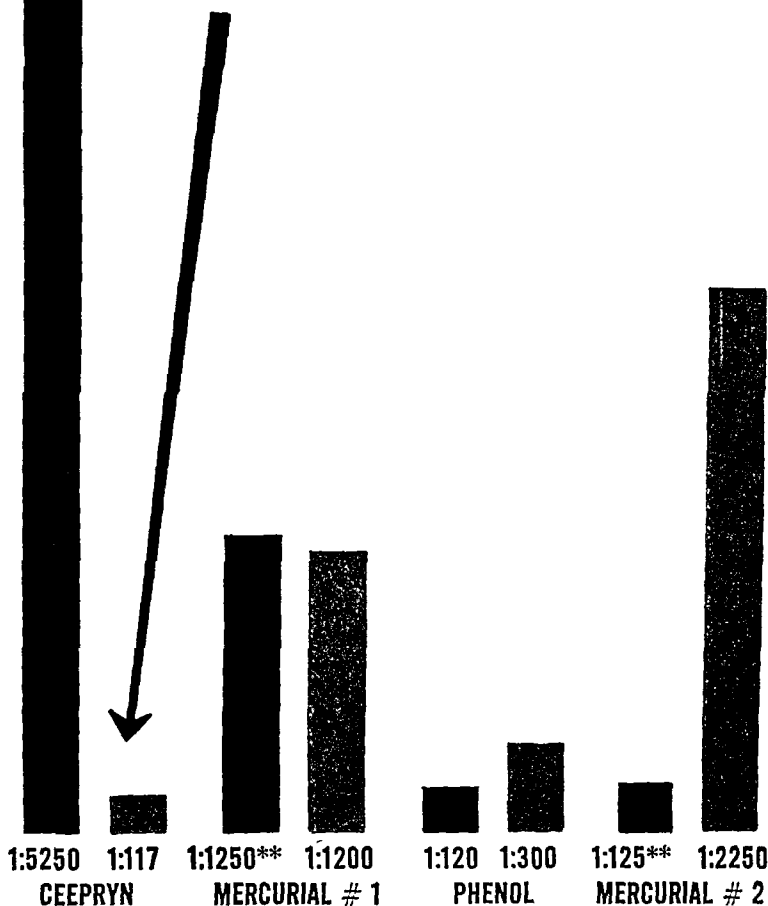
**Failed to kill

REFERENCE: Green, T. W., and Birkeland, J. M.: A method of determining the toxicity of disinfectants, J. Bact. 43:641 (1942).

Germicidal dilution against Staph. aureus in presence of serum (as determined by Salle method).

*"Critical Toxic Dilution" to tissue, as determined by inhibition of pulsation of chick embryo heart fragments.

*T*HE value of an antiseptic is determined by its ability to destroy bacteria effectively and leave the tissues unharmed. Ceepryn, a non-mercurial, non-phenolic compound, accomplishes this to a high degree. Its toxicity to tissues is almost negligible in comparison with its extremely potent bactericidal value.

Factors which recommend **CEEPRYN** *for general use:*

- Potent germicidal action in high dilution
- Effective against wide range of organisms
- Active in presence of serum
- Low surface tension—penetrating and detergent
- Low toxicity—no mercury, iodine or phenols
- Safe and non-irritating to tissue
- No interference with healing

CEEPRYN

Brand of Cetylpyridinium Chloride

3 DOSAGE FORMS, ALL REASONABLY PRICED

Ceepryn Tincture 1:200—4 oz., pints and gallons
Ceepryn Tincture 1:500—4 oz., pints and gallons
Ceepryn Aqueous Solution 1:1000—pints and gallons

Trade Mark "Ceepryn" Reg U S Pat. Off.

Write for sample and descriptive brochure

THE WM. S. MERRELL COMPANY

Founded 1828 • CINCINNATI, U.S.A.

PRE-
OPERATIVELY

White's

NEO MULTI-VI
CAPSULES
EACH CAPSULE CONTAINS:

Vitamin A	5,000 U.S.P. Units
Vitamin D	500 U.S.P. Units
Thiamine Hydrochloride, U.S.P.	1.5 mg.
(Vitamin B₁, 500 U.S.P. Units)	
Riboflavin	2.5 mg.
Pyridoxine Hydrochloride	1 mg.
(Vitamin B₆)	
Calcium Pantothenate	1 mg.
Nicotinamide	20 mg.
Ascorbic Acid, U.S.P.	50 mg.
(Vitamin C, 1,000 U.S.P. Units)	

POST-
OPERATIVELY

Centered around any surgical procedure are the factors which make the patient a better operative risk and reduce the post-operative hazards.

Since recent work has shown that various vitamin deficiencies may be the cause of such surgical difficulties as delayed wound healing and increased tendency to infection, it is rational to step up the vitamin intake during both the preoperative and the convalescent period.

White's Neo Multi-Vi Capsules offer valuable support to the diet of the surgical patient. One Neo Multi-Vi Capsule provides every major vitamin, including all those clinically established in amounts substantially greater than adult Basic Daily Requirements*—yet does not burden the patient with the cost of supplemental vitamins in amounts beyond his needs and perhaps even beyond his power to assimilate.

Supplied in bottles of 25, 100, 500 and 5000 capsules. White Laboratories, Inc., Newark, N. J.

*Promulgated in regulations of Food and Drug Administration, 1941.

White's PRESCRIPTION vitamins

LIPIODOL   in X-Ray Diagnosis

The introduction of Lipiodol considerably broadened the diagnostic value of roentgenography. During the past two decades, a vast bibliography has been accumulated. A large number of investigators have evolved many technics for its application. Through its use, many deviations from normal are diagnosed which otherwise would remain undetected.

Lipiodol in bronchography

Lipiodol in hysterosalpingography

Lipiodol in paranasal sinus visualization

Lipiodol has been instilled into virtually every cavity or viscus of the human organism. Containing 40 per cent iodine in firm chemical combination with poppyseed oil, it is highly radiopaque, casting a dense shadow on the roentgen film.

Lipiodol is widely used in the roentgenographic exploration of the paranasal sinuses, the bronchi, the uterine cavity and the fallopian tubes, the spinal subarachnoid space, joint spaces, and fistula and sinus tracts. It is virtually nonirritating, and if retained in situ, is slowly broken down and its iodine is excreted via the kidneys.

Physicians are invited to send for a complimentary copy of the brochure "The Applicability of Lipiodol in Roentgenography and the Technic of its Use," which describes in detail the many uses of Lipiodol.

Lipiodol in soft tissue sinuses

Lipiodol in fistula tracts

Lipiodol in roentgenexploration of empyema cavities

Lipiodol in sialography

Lipiodol in myelography

Editorial

OUR SURGEONS IN THE PRESENT WAR

PRESIDENTIAL ADDRESS
HENRY C. MARBLE, M.D.
BOSTON, MASSACHUSETTS

TWENTY-FIVE years ago, when the United States entered the first World War, the surgeons of this country were called upon to wrest themselves from their peace-time practices and join with their countrymen on the surgical staffs of our fighting forces. At that time we had had very little training for the task that was before us. Although the surgery of wounds is the oldest branch of medicine, the surgeons of our era had been so much concerned with abdominal, gynecological and cranial technics that little time had been given to the teaching of this ancient art. It may have been that the optimism of peace and the inability of most of us to imagine a world-wide war in a time of apparent enlightenment was responsible for this neglect.

When we were confronted with the emergency, we hurriedly adopted from the French and the English, who had been deep in this work for over two years, all that we believed to be good and we applied this borrowed knowledge in the care of our own wounded men. What we lacked in training and experience we tried to overcome by willingness and application of sound fundamentals. The end results of our efforts and the consequences of our failures are medical history.

After the Armistice we returned to our peace-time practices but not to our previous neglect of the study and teaching of the surgery of injuries. Realization came to the war surgeon that the problems of

159

war and peace are not foreign to one another; that the difference is only a change in location; the people and problems are the same.

During the past twenty years industry has expanded, the motor car has become popular, speed has increased and accidents are more numerous. Our hospital clinics are filled with the injured and wards are set aside for their care. The war surgeon continuing his interest in the surgery of trauma, offered his knowledge and experience to the younger staff members, and through research with them developed new medicines, means and methods.

And now another World War is upon us and thousands of young surgeons have again been called upon to care for the war wounded. Surely the surgeon going out today is far better trained, has a deeper knowledge and more mature judgment than his older brother of a quarter century ago. The soldier going into action today knows that he will be cared for by the finest trained and equipped corps of doctors that his country can give him and that, if he is wounded, his chances of recovery are greater than ever before. Knowing this he will do his job better.

The latest step in this educational trend is the organization of the American Association for the Surgery of Trauma, and its duty is clear. We must maintain a thinking membership to administer and direct; we must so plan our councils that we may receive, evaluate and judge all that is new in wound surgery. We must welcome all who are interested and qualified to the end that this knowledge may be spread to the profit of all. In war even more than in peace we have our work to do.

Officers of
THE AMERICAN ASSOCIATION
FOR THE SURGERY OF TRAUMA

President . . . . CHARLES S. VENABLE, M.D., Texas
President-Elect . . . . GROVER C. PENBERTHY, M.C., U.S.A., Michigan
Vice-President . . . . RALPH G. CAROTHERS, M.D., Ohio
Secretary . . . . GORDON M. MORRISON, M.D., Massachusetts
Treasurer . . . . ARTHUR R. METZ, M.D., Illinois
Recorder . . . . R. ARNOLD GRISWOLD, M.C., U.S.A., Kentucky
Acting Recorder . . . . . . W. L. ESTES, JR., M.D., Pennsylvania

Council

HENRY C. MARBLE, M.D., Massachusetts

FRASER B. GURD, M.D., Canada   CASPER F. HEGNER, M.D., Colorado
EDGAR L. GILCREEST, M.D., California   WILLIAM DARRACH, M.D., New York

Original Articles

SUPRACONDYLAR FRACTURES IN CHILDREN

ALEXANDER P. AITKEN, M.D.,
Junior Visiting Surgeon, Boston City Hospital
BOSTON, MASSACHUSETTS

LYMAN SMITH, M.D.
Formerly Resident Surgeon, Boston City Hospital
BARRINGTON, ILLINOIS

AND

CHARLES W. BLACKETT, M.D.
BOSTON, MASSACHUSETTS

ONE of the most difficult fractures to treat in childhood is the supracondylar fracture of the humerus. These fractures are often not only difficult to reduce, but are difficult to maintain in position. It is the general belief that accurate reduction in children is not essential for a good result. It is true that the functional end results of malalignment are generally very good, but it is also true that the cosmetic end results are often very poor. No patient can be expected to be satisfied with an excellent functional result if he also has a cubitus varus deformity. Our experience at the Boston City Hospital with the accepted forms of treatment has not been satisfactory. Recurrence of displacement has occurred despite accurate reduction and immobilization in flexion either by means of adhesive strapping or plaster splints. This has been particularly prone to occur in cases showing ulnar displacement with rotation of the distal fragment. Because of our dissatisfaction with our methods of treatment, we have made an end result study of fifty cases to determine the results of malalignment and to develop a more satisfactory method of treatment.

In this series of cases, the length of time elapsed between the dates of injury and examination, has not been less than three years and not more than seven years.

The original x-rays could not be obtained in one group of twelve cases. The end results of this group are as follows: Six cases are per-

fectly normal, functionally, cosmetically and by x-ray. Two cases show slight loss of the carrying angle, one a slight increase of the carrying angle and three a definite cubitus varus deformity.
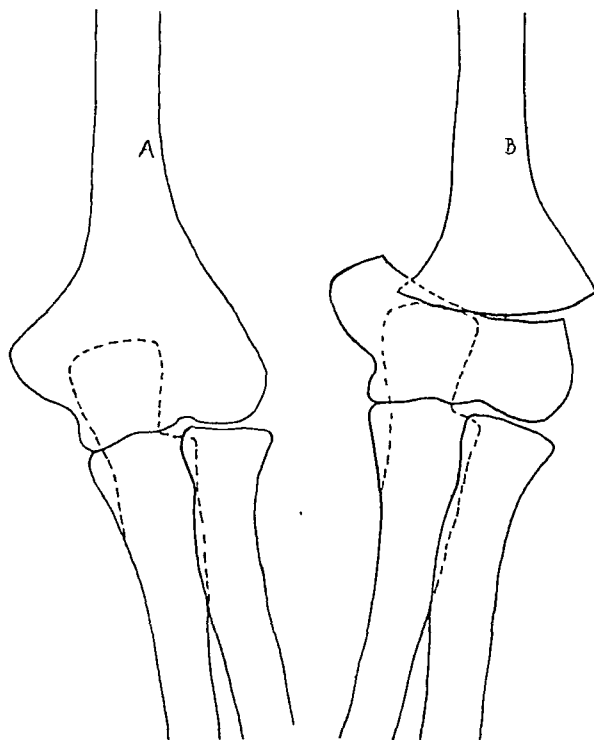
FIG. I. A, showing normal carrying angle of left elbow. B, showing loss of carrying angle by ulnar displacement of distal fragment.

The original x-rays in the remaining thirty-eight cases showed the following types of displacement: No displacement, five cases; posterior and ulnar displacement with rotation of the distal fragment, sixteen cases; posterior and radial displacement with rotation of the distal fragment, eleven cases; posterior displacement with rotation of the distal fragment but without lateral or medial displacement, four cases; anterior displacement, two cases.

The end results of the various types of displacement are as follows.

A. *Cases originally showing no displacement (five cases).*

The original position was maintained in all cases.   All now show no evidence of injury functionally, cosmetically or by x-ray.

B. *Ulnar and posterior displacement with rotation (sixteen cases).*

Excellent reduction was obtained and maintained in four cases. All now show excellent results. In nine cases, the displacement either

recurred or was incompletely reduced. All cases united with persistent ulnar and posterior displacement with rotation. All cases developed definite varus deformity. In two cases, the ulnar and

A   B
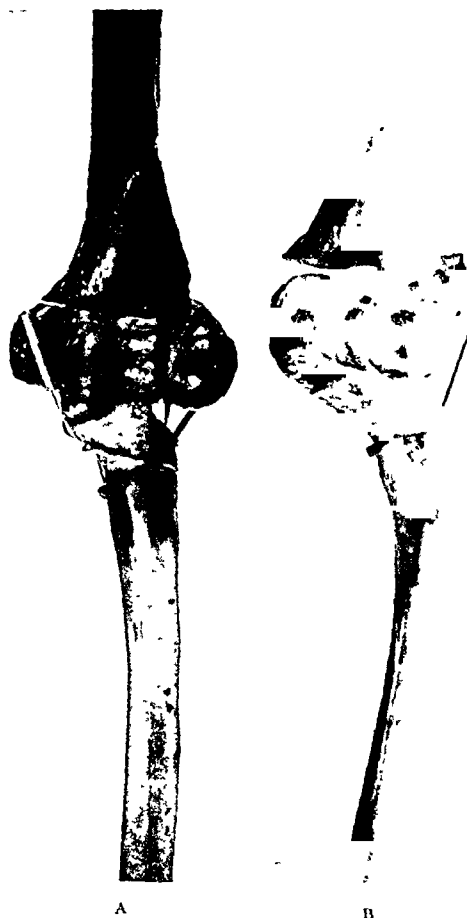
Fig. 2. A, showing normal carrying angle. B, shows loss of carrying angle due to rotation of distal fragment.

posterior displacements were corrected, but rotation of the distal fragment persisted. Both cases now show slight loss of the carrying angle.

One case had none of the components of displacement corrected, yet now shows an excellent result. Although x-rays failed to reveal involvement either of the trochlear or capitellar epiphysis by the fracture line, yet the trochlear epiphysis was apparently stimulated. The resulting overgrowth of the medial half of the shaft prevented the development of a varus deformity and brought about a normal

carrying angle. We have shown in previous publications that stimulation of an epiphysis by a remote fracture does occur. In this particular case we feel sure that the normal result was due to
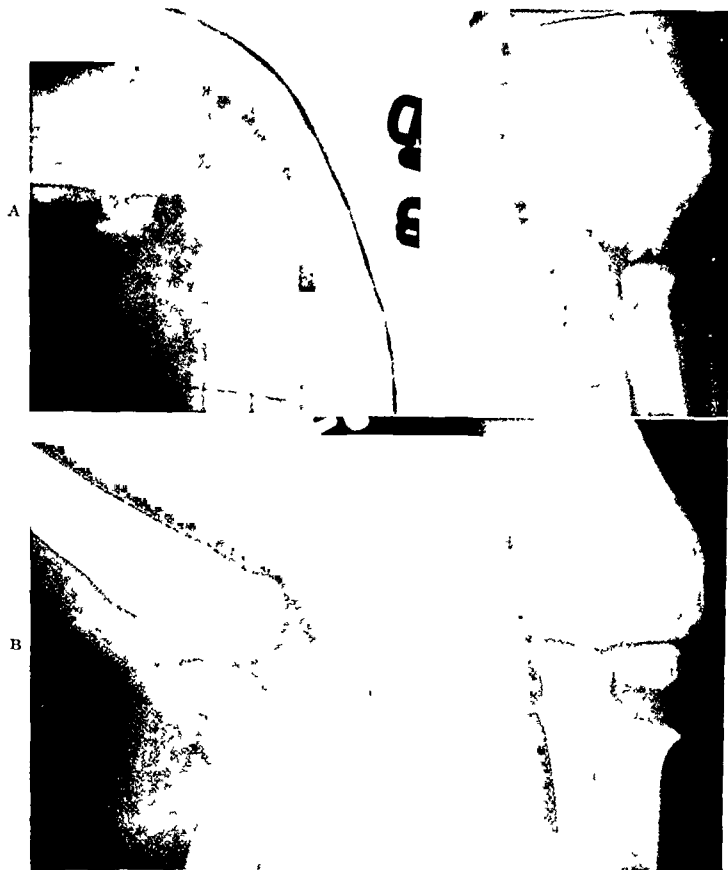
FIG. 3. A, case showing marked ulnar displacement with callus formation. B, same case, one year later, showing considerable varus deformity due entirely to the ulnar displacement.

stimulation of the trochlear epiphysis and not to retardation of the capitellar epiphysis.

C. *Radial and posterior displacement with rotation (eleven cases).*

An excellent reduction was obtained in only one case, which now shows an excellent result. Only partial reduction of all components was obtained in seven cases. All now show excellent results. In one case posterior displacement and rotation were corrected but radial displacement persisted. An increase in the carrying angle was the end result. Another case had only slight radial displacement but rather marked rotation and now shows a varus deformity of 5 de-

grees. In one case the displacement was corrected from radial displacement to one of ulnar displacement and now shows a marked varus deformity.

D. *Posterior displacement and rotation only* (*four cases*).

Two had excellent reductions and now show no evidence of their injury. One had correction of the posterior displacement but not of the rotation and now shows some loss of the carrying angle.

One had slight persistent posterior displacement with considerable rotation and now shows complete loss of the carrying angle.

E. *Anterior displacement* (*two cases*).

One case showing radial displacement was not completely reduced but shows now an excellent result. One case showing ulnar displacement was not completely reduced and developed a varus deformity. This case also showed a vertical T-fracture which extended into the capitellar epiphysis. Contrary to what might have been expected, this epiphysis did not show retardation of growth but was stimulated by the fracture as the deformity has increased over a six-year period.

In an excellent article on the end results of supracondylar fracture, Siris presented several cases in which either the trochlear or capitellar epiphyses were involved in the fracture. In his cases the involved epiphyses showed retardation of growth with resulting deformity. Our experience with fractures of the epiphyses has been similar to that of Siris. Stimulation of the epiphysis by the fracture, as in the above cases, is very unusual. Brewster and Karp believe that the varus deformity in such fractures is due to stimulation of the capitellar epiphysis. Such stimulation probably does occur in some cases. Deformity, then, can result either from stimulation or retardation of the epiphyses whether the epiphyses are directly involved in the fracture line or not. However, in the great majority of cases deformity is not due to epiphyseal involvement but to malposition of the fragments, particularly in those cases with persistent ulnar displacement and rotation. In all but one of our cases in which such displacements were left unreduced, either gross varus deformity or loss of the carrying angle resulted.

It is to be remembered that the normal carrying angle of valgus is due to the normal obliquity of the distal end of the humerus. Any change in this plane will, of course, change the carrying angle. Displacement of the distal fragment ulnarly causes the articular surface to face medially and a position of varus is the result. (Figs. 1, 3 and 4.)

FIG. 4. For descriptive legend see opposite page.

Normally the carrying angle is obliterated on pronation of the forearm. When the medial end of the distal fragment rotates posteriorly, the forearm follows the distal fragment, thus the distal fragment and forearm become, so as to speak, pronated on the proximal fragment. (Fig. 2.) By such rotation, the carrying angle becomes lessened or obliterated. In this respect it is interesting to note: (1) In two cases in which the ulnar displacement had been corrected but in which rotation persisted, a loss of the carrying angle was the result in each case. (2) In seven cases in which there had been persistent radial displacement and in which we might have expected an increase in the carrying angle, rotation has also been present which compensated for the radial displacement and normal carrying angles have resulted.

In order to prevent deformity it is, therefore, imperative to reduce ulnar displacement and rotation of the distal fragment, and to maintain such reduction. Maintenance of such a reduction we have found difficult with the accepted methods of treatment. After reduction and the application of either adhesive or a plaster cast, it is the customary practice to place the forearm across the chest. In so doing the forearm and the distal fragment are internally rotated and it is assumed, once reduction has been obtained, that the proximal fragment also undergoes such rotation. If the fragments are locked, especially in acute flexion, such rotation of the proximal fragment will occur. However, such flexion cannot always be obtained because of swelling or interference of the radial pulse. In the absence of such flexion, the obliquity of the fracture line and the narrowness of the humeral shaft at the fracture site render locking difficult. In this event, when the forearm is placed across the chest, only the distal fragment rotates and the position obtained by reduction is lost. With the recurrence of rotation, backward and ulnar displacement can easily occur, especially if the immobilizing agent fails to

FIG. 4. A, case showing original ulnar displacement in anteroposterior view. B, lateral view of same case. Note in attempting to get this view only the distal fragment rotated. The position of the shaft remains the same in both views. Similar rotation occurs, after reduction, in unlocked fractures when the forearm is placed across the chest. c, same case (anteroposterior view) two years later with opposite normal elbow. These views were taken with the condyles of both humeri lying in the same plane. The fracture has healed with almost 90 degrees rotation as in Figure 4B. Note views of condyles are anteroposterior while that of the shaft is lateral. D, same case, both elbows in lateral view. Note view of condyles is lateral while that of shaft is anteroposterior. The deformity in this case was largely due to this extreme rotation.

FIG. 5.
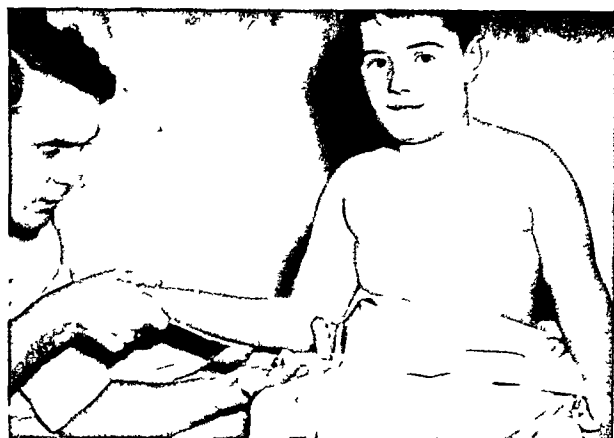
FIG. 6.
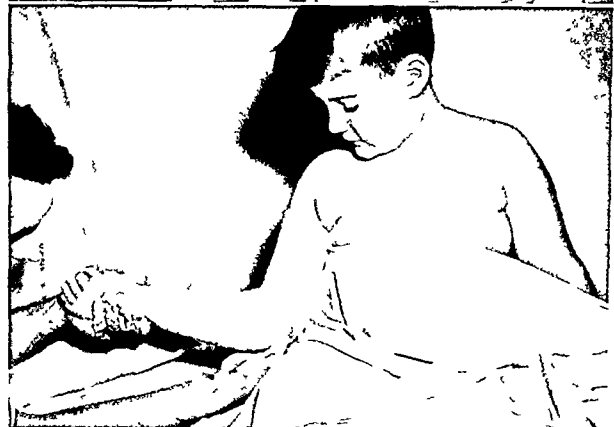
FIG. 7.

FIG. 5. Reduction by Cotton method of external rotation of
the entire upper extremity with the forearm supinated.

FIG. 6. Cast applied to arm with elbow at right angles. This
position is used only when swelling and interference of
radial pulse allow no further flexion.

FIG. 7. Chest swathe of plaster applied holding distal frag-
ment and forearm in external rotation. When further
flexion is possible, such complete external rotation is
usually not necessary.

give proper support for the weight of the forearm. We believe this practice of placing the forearm across the chest, to be the chief reason for our failures to maintain position.

We have been able to maintain position much more satisfactorily since the following method was adopted. Reduction is obtained by the method described by Cotton, namely, outward rotation of the entire arm. (Fig. 5.) By this procedure the elbow is flexed at a right angle and the forearm is abducted which tends to rotate the distal fragment externally. We also believe the forearm should be supinated. By so doing we are of the opinion that tension is placed on the pronator radii teres which further prevents the internal condyle from rotating backward. With the extremity thus rotated outward, the elbow is flexed as much as possible, depending upon the amount of swelling and the quality of the radial pulse. Plaster strips are then applied to the arm and allowed to set partially. A plaster swathe is then placed around the chest and incorporated in the plaster about the arm. (Figs. 6 and 7.) In this manner the fragments are locked in flexion and external rotation and the chances of recurrence are definitely less. After the adoption of this method, we found a similar procedure had been described by Eliason. He believed, in employing the acutely flexed position, that the hand should be carried to the shoulder of the injured side to prevent recurrence of rotation. He does not externally rotate the arm as we do, and in cases when acute flexion can be accomplished, his method should be satisfactory. When acute flexion cannot be maintained, further external rotation should be employed.

Some other interesting facts learned from this study are as follows:

There were two cases of so-called myositis ossificans. Both cases were slow in recovering function but all evidence of calcification and limitation of motion disappeared within two years from the date of injury.

Nerve involvement was seen in four cases. The radial nerve was involved in three instances, the ulnar in one. Both cases of anterior displacement showed nerve involvement, one radial and one ulnar. In all cases complete recovery occurred from six to ten weeks after injury.

There was but one case showing circulatory embarrassment. This was placed in traction for a few days and then reduced with an excellent end result.

Although the functional end results were excellent, some cases did show changes in the range of motion. In three cases there was a

loss of flexion of from ten to twenty degrees. Four cases showed a loss of extension of from ten to twenty degrees. In six instances there was an increase in the range of extension of from ten to twenty-five degrees without loss of flexion. Three of these cases have varus deformity.

In summarizing the end results of fifty cases of supracondylar fracture we find almost uniform excellent function. Cosmetically, there were twenty-seven excellent end results. There were two cases of increased carrying angle and three cases of decreased carrying angle. There were three cases of complete loss of the carrying angle. Although the above eight cases show change or loss in the carrying angle, they cannot be classified as showing poor cosmetic results. There were, however, fifteen cases of definite varus deformity which can only be classified as poor cosmetic results.

CONCLUSION

Supracondylar fractures with radial displacement and rotation of the distal fragment show excellent function and cosmetic end results, although complete reduction may not be obtained.

Varus deformity is due to ulnar displacement plus backward or internal rotation of the distal fragment. Persistence of either condition may lead to loss of the carrying angle or varus deformity. Persistence of both conditions leads almost invariably to the poor cosmetic result of cubitus varus.

Recurrence of deformity we believe is due to the practice of placing the forearm across the chest.

A method of immobilization is described which it is hoped will give better results.

There seems little excuse for poor cosmetic results in this type of fracture which we believe are to be universally found. The methods of reduction and immobilization were described by Cotton and Eliason eighteen years ago. The popular belief that accurate reduction is not essential to a good result is condemned.

REFERENCES

1. COTTON, FREDERICK J. *Dislocations and Joint Fractures*. 2nd ed. Philadelphia, 1924. W. B. Saunders Co.
2. ELIASON. *J. A. M. A.*, June 14, 1924.
3. SIRIS, I. E. Supracondylar fractures of the humerus. *Surg., Gynec. & Obst.*, 68: 201, 1939.
4. BREWSTER, A. H. and KARP, M. Fractures in the region of the elbow in children, *Surg., Gynec. & Obst.*, 71: 643, 1940.

5. Thorndike and Dimmler. Fractures of the forearm and elbow in children. *New England J. Med.*, 225: 475, 1941.

6. Watson-Jones. Fractures and Other Bone and Joint Injuries. 2nd ed. Baltimore, 1941. Williams & Wilkins.

7. Aitken, A. P. Epiphyseal Injuries in the Treatment of Fractures. Charles Scudder. 11th ed. Philadelphia, 1938. W. B. Saunders Co.

8. Aitken, A. P. Overgrowth of the femoral shaft following fracture. *J. Bone & Joint Surg.*, 21: 334, 1939; *Am. J. Surg.*, 49: 147, 1940.

DISCUSSION

Fred H. Smith (New York City): I would like to ask Dr. Aitken what he has been doing for the supracondylar fractures with marked circulatory disturbance in which the displacement was great and the soft tissue damage proportionately increased. In New York, we have been using the Kirschner wire and putting the arm in traction, hanging the elbow above the patient with the patient lying supine in bed. By that means, we are able to keep the elbow elevated. There is no obstruction to either the venous or lymphatic return, the patient is kept quiet and it is practically a painless procedure, and we are also able to control rotation of that distal fragment. If we find in subsequent check-up by x-ray that there is rotation, we can turn the forearm this way or that way and control that distal fragment.

Out of 218 cases in a nine-year period from 1929 to 1937, inclusive, we have used it in twenty cases, that is, just under 10 per cent, and out of that twenty, eight patients have had essentially perfect results. The remaining thirteen have had what would be called excellent results. A few of those had some loss of carrying angle and some loss of flexion. Some had one or the other, some had both, but the function was essentially perfect and they could do everything they wanted to.

As far as complications are concerned, we had three postoperative wristdrops, all of which cleared up subsequently. The only other complications were mild pressure sores at the side of the wire and no serious osteomyelitis.

Out of the entire series there was no case of Volkmann's paralysis. One patient came in with a beginning Volkmann's which had been going on only about five hours from the time of the onset. He had loss of pulse, some loss of nerve function and the fingers were cold. We put in the Kirschner wire and the impending Volkmann's immediately cleared up and he never developed any further complication.

Alexander P. Aitken (closing): I do not believe we have adopted any one procedure for the handling of these circulatory disturbances. In this one particular case, the arm was placed in traction with adhesive along the forearm and suspended from a Balkan frame. If there is any question of circulatory embarrassment, with an absent pulse, I know some men (and I have done it myself), have gone in and split the bicipital fascia. Other men have used Kirschner traction. We have not adopted any one method for handling it. It varies with the man who happens to get the case.

POSTOPERATIVE PAROTITIS WITH
RADIATION TREATMENT

DANIEL C. PATTERSON, M.D.

BRIDGEPORT, CONNECTICUT

PAROTITIS, when a complication of surgical procedures, is a very serious condition. Statistics gathered previous to irradiation therapy show a mortality rate of over 50 per cent. Blair and Padgett, in 1923, reported on thirteen cases of postoperative parotitis with seven deaths. Fortunately, it is a rather rare occurrence, the incidence being given as anywhere from one case in one thousand to one case in two thousand operations. Rankin and Palmer, in 1930, surveying a four year period at the Mayo Clinic, found twenty cases in 2,700 operations on the colon and rectum, a ratio of 1 to 135. They concluded there was an increased incidence with the increased development of surgery of the large bowel. They could not explain why. In 1937, Maurer reported thirty-three cases in 35,000 operations at the Munich Surgical Clinic. He also reported fourteen deaths in these thirty-three cases. I do not believe the high mortality rate of surgical mumps has been generally appreciated by the profession.

Numerous theories have been advanced to account for its occurrence, but none of them is fully satisfactory. In 1887, Stephen Paget investigated the records of one hundred cases and concluded that in those cases in which it followed operations on the genital organs it was due to close relationship between these organs and the parotid gland. Dr. Howard Kelley writes: "It is my conviction that postoperative parotitis is due to thumbing of the parotid gland by the anesthetist." It might be pointed out that the disease is not partial to operations upon the genital tract, and it has followed operations under local anesthesia in which there was no manipulation about the patient's jaw. Debilitation, dehydration and foci of infection in the oral and nasal cavities are generally mentioned as contributing factors, but great numbers of patients with these unfavorable conditions are operated upon daily and parotitis remains a rare complication. Moreover, as Rost points out, "It is almost never observed as a result of stomatitis."

Postoperative parotitis differs widely from simple parotitis or mumps. The latter condition, which is a virus disease, has a long

incubation period, no tendency to suppuration, and a negligible mortality; whereas surgical mumps has a short incubation period, does tend to suppurate, and has a high mortality. The theory that it was of embolic origin or due to pyemia has little support at present. Pyemic parotitis does occur in the presence of sepsis, but surgical mumps may occur in the absence of any apparent infection. In pyemic parotitis it has been shown that the infection begins around the vessels in the parotid gland, while in postoperative parotitis it begins around the ducts. Rost says, "There is no difference in the anatomical picture between the ascending and the hemotogenous routes of infection." The staphylococcus is the organism most frequently involved. This is true even though the patient may be suffering from an infection due to another bacteria.

The disease may follow any type of operation whether septic or not. I remember one fatal case that followed the dilating of a urethral stricture. It is usually ushered in with sudden high fever and malaise within a week after operation. The infected gland early shows swelling, and the diagnosis should be easily made. It is most important that the condition be recognized in its early stage, for a successful result demands early treatment. Nurses and interns should be instructed to be on the look out for the first signs or symptoms.

Many forms of therapy have been advocated in the past. Local applications, mouth washes, catheterization of Stenson's duct, use of vaccines, and wide incisions when suppuration is present, have been the general treatments.

Leithauser and Cantor treated twenty-three patients with large doses of Lugol solution. Twenty minims were given every three hours, day and night. Fulminating cases were given 1 to 2 dr. in 1,000 cc. of solution intravenously. Dosage was decreased as improvement took place. They advised aspiration with a large needle instead of incision if suppuration occurred. This treatment might be worth trying if radiation is not available.

The sulfonamides are probably of value. Walker and Allen report a case of bilateral postoperative parotitis treated with 10 gr. every six hours, but radiation was also used in their case. R. E. Smith reports quick recovery from a case of ordinary mumps under sulfanilamide therapy.

Bowing and Fricke reported on the use of radium in 184 cases at the Mayo Clinic. Thirty-two of these cases had bilateral involvement, and the mortality was 40 per cent. Of the other 152 patients, 19 per cent died. The authors added that 6 per cent died of parotitis

alone. They advised radium in moderate doses, with local treatment of warm, moist dressings, and incision when suppuration occurred.

In 1936, Dr. Fred M. Hodges, of Richmond, Virginia, reported that he had successfully treated a fairly large number of patients with acute, subacute and chronic parotitis since 1924 with roentgen therapy. He stated that the majority of surgeons were unaware of the value of irradiation in this field. He advised three to five treatments daily, or every other day, using 125 to 170 kilovolts, with about 125 to 200 R units. Dr. Hodges deserves the credit for bringing this form of treatment to the profession.

Following the method of Hodges, Latchmore and associates reported eleven cases of postoperative suppurative parotitis in which the patients were treated by x-ray therapy. Complete resolution occurred in six, two recovered after incision of a local abscess, and resolution was taking place in one when the patient died of another cause. Two patients treated late, that is, ten days after the swelling appeared, died.

Five cases of postoperative parotitis occurred on my service at the Bridgeport Hospital between May, 1940, and August, 1941. These patients were treated by radiation therapy under the direction of Doctors Parmelee and Lockhart, and the results are the reason for this report.

CASE REPORTS

CASE I. Mrs. M. L., age sixty-five, was operated upon May 27, 1940. A cyst of the right kidney was removed. On May 30, the patient complained of soreness in the left side of the face. The left parotid gland was swollen and tender; her temperature was 102°F. She was given 450 R units in the course of five days. The swollen gland subsided and the temperature was normal by June 9.

CASE II. Mrs. M. G., age eighty-five, was operated upon June 28, 1940, when a colostomy for intestinal obstruction was performed. On July 14, the left parotid gland was swollen and tender; the patient's temperature was elevated. X-ray treatment was started, and she was given 150 R units every other day for three doses. Her temperature was normal on July 22, and she went home on July 24.

CASE III. Mary F., age sixty-five, had a gastric resection for carcinoma on November 12, 1940. On November 16, the patient was listless and drowsy; her temperature was 101°F. At 8 A.M. it was noted there was a large swelling of the right parotid gland. At 9:30 A.M. she received her first radiation treatment of 195 R units. On November 17, the patient was given a second treatment, and two days later a third dose. The gland subsided

promptly and her temperature was normal by November 27. She was discharged one week later.

CASE IV. Mrs. E. W., age fifty-eight, had a cholecystectomy and freeing of adhesions performed on July 24, 1941, and also cecostomy for obstruction (inflammatory) of the colon. On August 4, the right parotid gland was swollen; her temperature was 103°F., and leucocyte count 33,000, with 91 per cent polymorphonuclears. X-ray treatment was started at once and repeated on August 5. The temperature dropped and the swelling of the parotid subsided in a few days. On August 14, there was a recurrence of the parotitis and x-ray treatments were again given on August 14, 15 and 16. The response was prompt and the patient proceeded toward a good recovery.

CASE V. Miss D. H., age twenty, was operated upon July 28, 1941, for acute purulent appendicitis. On July 31, there was swelling and tenderness of the left parotid gland. The patient had a chill and temperature of 103°F. X-ray treatment was started within a few hours after the swollen gland was first noticed. She received two treatments on successive days. By August 4, the temperature was normal and the parotid swelling much less. The patient had no further trouble.

I am unable to explain why these cases occurred. In only one instance were two of the patients in the hospital at the same time, and all were in different wards. Three had nitrous oxide ether anesthesia by machine. One had open drop ether after gas induction, and one local novocain infiltration. Different anesthetists were involved, as well as various doctors and nurses. There had been no similar cases for a long time previously, and there has been none since on my service.

Aside from radiation, local treatment only was employed in these cases. The radiation was begun early, and in no case did suppuration occur, although it appeared imminent in some of the glands. The prompt recoveries and absence of suppuration indicate that x-ray radiation is a very valuable agent for the treatment of postoperative parotitis, and we believe that the splendid results obtained in these cases were due to the early application of radiation.

As prophylactic measures, no elective surgery should be done when foci of infection are present in the nose, mouth or throat. Debilitated and dehydrated patients should receive proper preoperative treatment.

SUMMARY

1. Postoperative parotitis is a very serious complication.
2. X-ray radiation is a most effective treatment.

3. Radiation should be started early in the disease to prevent suppuration.

4. More attention should be paid to preoperative preparation of surgical patients.

REFERENCES

1. BLAIR, V. P. and PADGETT, E. C. Pyogenic infection of the parotid gland and ducts. *Arch. Surg.*, July, 1923.
2. RANKIN, F. W. and PALMER, B. M. Treatment of postoperative parotitis. *Ann. Surg.*, Dec., 1930.
3. MAURER. Munich Surgical Clinic. Quoted in Year Book of Surgery, 1938, p. 184.
4. PAGET, STEPHEN. Parotitis after injury or disease of the abdomen. *Brit. M. J.*, Feb. 12, 1887.
5. KELLY, H. Operative Gynecology. New York, 1909. D. Appleton & Co.
6. ROST, FRANZ. Pathological Physiology of Surgical Diseases. Philadelphia, 1923. P. Blakistons & Co.
7. LEITHAUSER and CANTOR. Massive doses of Lugol's solution in acute secondary parotitis. *Ann. Surg.*, vol. 3, p. 650.
8. WALKER, M. A. and ALLEN, L. G. Post operative parotitis; recovery after sulfanilamide and roentgen therapy. *J. Kansas State Med. Soc.*, July, 1939.
9. BOWING, H. H. and FRICKE, R. E. Radium treatment of post operative parotitis. *Radiology*, Jan., 1936.
10. HODGES, FRED M. Roentgen therapy of certain infections. *Roentgenology*, Feb., 1936.

PERIPHERAL CIRCULATION IN RELATION TO TRAUMA*

WITH SPECIAL REFERENCE TO THROMBOSIS AND EMBOLISM

ARTHUR W. ALLEN, M.D.

Chief of the East Surgical Service, Massachusetts General Hospital; Lecturer in Surgery, Harvard Medical School

BOSTON, MASSACHUSETTS

WHEN I was first approached on the subject of peripheral circulation in relationship to injury, I had in mind that I was supposed to deal with injured blood vessels—the effect of healing of tissues distal to such lesions, etc. I have been informed, however, that Dr. Marble's idea was to have me discuss the question of thrombosis and embolism, which had interested us in this Hospital so much during the past year. I very much regret that one of your members, Dr. Henry H. Faxon, is not here to present this phase of the subject since he[1] and Dr. Claude E. Welch and Dr. C. E. McGahey have made a special study of this situation and have sent in for publication an excellent article on the subject. Since all of these men are now with the Military Forces, I will endeavor to give you a résume of our experience in this field.

Although thrombosis followed by embolism is not as common in the average patient who suffers an injury, as it is following many major surgical procedures, there are instances that make us realize that one must be on the alert for this possibility in all patients regardless of their type of illness or injury. For some time, it has been the policy of our X-ray Department to interpret certain signs in the chest as due to infarct with great accuracy. In addition to this, the Pathological Department have demonstrated that almost all fatal pulmonary emboli, which we have encountered, have originated in the region of the popliteal vein. When such a thrombus propagates in both directions, the long column of the femoral vein can produce an enormous clot and one that, when it breaks loose in its entirety, will very often completely occlude the pulmonary artery.

Several years ago, Dr. John Homans[2] of Boston read a paper before the New England Surgical Society advocating the ligation and division of the femoral or the iliac vein, following repeated sub-

lethal infarcts in patients who had a low-grade inflammatory or mechanical condition in the vein producing thrombosis. Although this was an interesting observation that Dr. Homans reported, three or four cases at that time, believing that he had perhaps prevented fatal embolus under these circumstances, I doubt if there was any widespread attention paid to this suggestion. Actually, it was two or three years afterward before I found a case that I thought fitted into his group and on whom I was willing to do a high ligation. Gradually, there has come about a much more radical attitude about the entire situation.

First of all, one must get over the feeling that interruption of the femoral or even the iliac vein will produce severe invalidism. There occurs some swelling in the extremity after ligation but this is not disabling; it lasts for varying periods of time, but rarely handicaps the patient in his usual routine of life. The disability, in fact, is so slight that our internist colleagues believe that it is entirely minor as compared with the risk of waiting to see whether the patient would develop a more massive embolus. It only takes an instance or two in a man's experience of fatal embolus in an otherwise healthy patient to make him feel considerably more radical about this whole matter.

Fatal pulmonary embolus occurred in this Hospital prior to Dr. Lincoln Davis' study[3] of fifteen years ago at the rate of three deaths in every thousand patients who had been subjected to a major surgical operation.

After this, various methods of prevention of phlebitis were instituted, such as elevation of the foot of the bed, bicycle exercises of the legs, frequent turnings, etc. These methods, with perhaps better attention to the handling of tissue, the elimination of pressure bandages over large vessels, have (according to a recent study by Drs. Welch and Faxon[4] on patients in this same Institution) reduced the number of fatal emboli to about one in a thousand operations.

At first, it seems as though we might be able to pick up the early signs of phlebothrombosis or thrombophlebitis by a routine, bi-daily, careful inspection of the extremities of these postoperative cases. This is done by the resident staff religiously and frequently enough early signs can be noted, although these may be minimal. It is not at all uncommon now to have these signs picked up before the patient is aware of any disturbance or before the chart indicates that there is this complication impending. These examinations con-

sist in an observation of the extremities, both being uncovered; the question of whether the superficial veins are filled or empty; whether there is any swelling by actual measurements; whether there is any tenderness in the calf of the leg, and whether or not there is a positive Homans' sign.

This last sign is by far and above the most helpful of all and is determined by the following method: With the patient's leg in extension, the palm of the left hand is placed gently on the patella; the right hand grasps the distal segment of the foot, which is gently but forcibly dorsiflexed. If during this maneuver, the patient is perfectly comfortable, one can feel reasonably assured that the veins in the popliteal space are not involved in any pathological process. If, on the other hand, the patient complains of discomfort in the calf of the leg or in the popliteal space when this maneuver is done, one can then be reasonably sure that future developments will reveal a thrombosis in the vein. We do pay considerable attention to superficial phlebitis when it develops and believe that, when it is discovered, ligation at the foramen ovale of the long saphenous vein is indicated. This, we believe, has perhaps prevented a good many instances of deep phlebitis and has made it possible to shorten the convalescence of these patients tremendously.

Although we see a number of cases that certainly fall into the group which Dr. Alton Cchsner[5] has called phlebothrombosis, we have here in this community a larger number of true inflammatory thromboses of the vein than they seem to have in New Orleans. It is probably due to the fact that we have more lowered resistance due to our climate, etc., than they find in the deep South. It is a well known fact that we have more postoperative pulmonary complications in this region than they have in warmer areas. This we believe is due to the same difference in climactic conditions.

In the recent study, it was found that 95 per cent of all of the fatal emboli occurred in people who had absolutely no sign whatever of a previous thrombosis. They had not complained of any discomfort in their legs, they had no change in pulse, temperature, or respiration, and undoubtedly most of these should be classified as phlebothromboses. In these patients, the pathologist invariably found the origin of the embolus to be in the deep veins of the leg.

Further analysis showed that in the patients between twenty-five and forty-five years of age, there were very few deaths, although there were a good many infarcts in the lungs in this group. Before we began doing ligations, these were all treated conservatively and very

few of them died. In the older group beyond the age of forty-five, the number of fatal emboli was very much greater; the tendency increases as the patient grows older, so that we began ligating the femoral vein in the older age group with greater frequency and with more feeling of satisfaction than we now have when any patient of any age group has any indication of phlebothrombosis or thrombophlebitis, since such a patient is subjected to ligation without further ado.

It was thought that perhaps we could be intelligent and scientific about selecting these patients for vein ligations. Venography is now quite a well established method of outlining the veins and it gradually became used in the study of these patients. It was believed at the time that Drs. Faxon, Welch and McGahey sent their paper in for publication, that perhaps venography should be done pretty often in a doubtful or borderline case. Although venography is still done, I wish to say that we are not quite so sure that it is a completely reliable test and we know, with the X-ray Department shorthanded as they are, that it puts an extra burden on them which perhaps in the future we may usually avoid. The method of venography is well described in the article referred to above and when such a test is definitely positive, one is greatly assured that ligation is indicated. If, on the other hand, it is negative, one may feel with a clear conscience that the veins need not be ligated. The difficulty with the method is that in some instances the vein has appeared normal and yet the patient has had an embolus; and some of the veins that have been explored and ligated in the presence of a normal venogram have shown thrombosis in the vein. On the other hand, some of the venograms, that have shown no filling whatever in the deep veins, have misled us in that at operation the vein has been found completely free of any thrombus.

When multiple emboli have occurred, the patient may well develop considerable difficulty in his chest which continues even after vein ligation. In instances in which veins that have appeared to be normal have been ligated, the infarcts have continued always sublethal, leading one to assume that these infarcts may well have come from the neighborhood of a wound and would never have proved fatal under any circumstances. Such patients sometimes are helped by heparin, although our experience with heparin here has not been as spectacular as it has been in Toronto. So far we have had no experience with dicoumarin and this may eventually prove to be a tremendous help in this field.

There is one very definite feature that I think should be stressed and that is the ligation of both femoral veins rather than the one that appears to be involved. We have expected the misfortune of ligating the vein on the side that was obviously involved with thrombophlebitis only to have the patient succumb to fatal pulmonary embolus, and to find at autopsy that the thrombus that had produced the embolus had come from the supposed normal side. Therefore, there is some argument that if one ligates the obviously involved vein, one must then very seriously consider ligating the supposed normal side. This bilateral ligation is being carried out here now with more and more frequency.

The technic of the operation is extremely simple: Under local anesthesia guided by the pulsation of the femoral artery, one makes a $2\frac{1}{2}$ to 3 inch incision over the course of the vein, starting in the crease of the groin. The long saphenous vein is investigated and if this is found not to be thrombosed and not varicosed, it is left alone. The femoral vein is exposed in the short space that is free below the profunda femoris. One can then pass two chromic catgut ligatures under the vein, leaving them untied but held by hemostats so that the bleeding can be controlled after the vein is opened. If one opens the vein transversely through half its diameter, leaving the posterior third or half of the vein intact, the manipulation is almost as easy as if the vein was completely divided and the control of bleeding from the vein, by holding up on the previously placed catgut ligatures, simplifies the procedure. After opening the vein, clots will exude from it and by using a glass-tipped Trendelenburg suction apparatus or a simple glass drinking tube, one can with suction pull the clot from the region of the iliac vein. After one has got all of this clot out, there should be free bleeding from the proximal segment. This bleeding is kept under control then by the elevation of the upper ligature. Then clot is milked and sucked from the distal segment of the vein, removing as much of it as possible. Frequently enough, clots from above and below put together may measure 16 or 18 inches in length. If one succeeds in getting the clot from the lower segment of the vein so that free bleeding takes place from below, the resulting edema from the ligation will be greatly diminished and the convalescence thereby will be shortened. After removing the clot from the vein, one simply ties the ligatures previously placed and then transfixes the vein with a cotton or silk suture distal to the catgut tie in order to insure closure of the vein in this region. One may leave the posterior half or third of the vein intact, thereby ligating the vein

more or less in continuity, or divide it. It seems to make no difference which method is adopted. We have reason to suppose that in time nature reëstablishes circulation through this tied-off vein, as we have venograms that would seem to prove that this actually did occur.

After ligation other things being equal as regards difficulty from the infarcts in the chest or the operative wound originally made, one may with perfect impunity make these patients ambulatory just as soon as their temperature is normal. It is a great comfort to be able to get these people out of bed at the end of forty-eight to seventy-two hours and have them walk about. They use at first an ace bandage on the lower extremity from the foot to the knee and often take this home with them but rarely use it for more than three or four weeks. The small amount of swelling which occurs after this time is so negligible and so unimportant from their standpoint that they prefer not to bother with the bandages. We have noted that there was some tendency for superficial veins to increase in size, temporarily at least, following the ligation of the femoral vein itself.

I am perfectly certain that time will finally settle the matter in a little more satisfactory fashion than it appears to be today. At the moment, I know we are ligating a great many veins that would not be the source of a fatal pulmonary embolism. I would suppose that perhaps we may save six lives out of each hundred patients on whom we do vein ligations. This is a definitely worth while salvage but with more experience and a more careful analysis, perhaps more useful standards of indications, we may be able to select these patients better and reduce the number of ligations with a consequently higher ratio of rescues. At the moment, it is difficult to persuade our medical colleagues that every infarct of the chest should not have immediate bilateral femoral ligation. We are at a loss to know how to answer this other than to comply with their request because, in the first place, we must admit that this is not a serious operation; it does not produce a serious handicap to the patient afterward; and, of course, at the moment we have no way of telling whether or not that particular patient would develop a massive embolus if his veins were left intact. There are many problems to be settled and one, I am sure, is how can we tell whether the infarct has not come from the heart, from the operative site, or from some smaller vein in the body other than the ones that are large enough to produce the amount of thrombus necessary to occlude the pulmonary artery completely. We believe, in time, that many of these questions will be solved; we plan a further analysis of our experience which is now getting greater

day by day. I think it is probably safe to say that at this time we have ligated already in this Hospital approximately 150 veins and this would probably represent about 125 patients. At the rate we are going, it will not be long before our experience will be very large. We know that it is an operation that can be done simply and easily; it takes almost nothing out of a very ill patient. It requires twenty to thirty minutes of time for each side, and the results in some instances are so spectacular that we believe it well worth while. In addition to the prevention of fatal embolus in the cases of thrombophlebitis, we find that the swelling and the pain resolves very much more quickly following the opening of the vein, the removal of the clot, and the ligation of the vein, than it would under ordinary circumstances. Patients, who used to remain in bed for six weeks to recover from a thrombophlebitis, would often be able to leave the hospital under these circumstances within a week.

REFERENCES

1. WELCH, CLAUDE E., FAXON, HENRY II. and McGAHEY, C. E. The application of phlebography to the therapy of thrombosis and embolism. *Surgery*, 12: 163–183, 1942.
2. HOMANS, JOHN. Exploration and division of the femoral and iliac veins in the treatment of thrombophlebitis of the leg. *New England J. Med.*, 224: 179, 1941.
3. DAVIS, LINCOLN. Personal communications re. chapter by Allen, A. W. Nelson's Loose-Leaf Surgery on Vascular Disease, July 1931.
4. WELCH, C. E. and FAXON, H. II. Thrombophlebitis and pulmonary embolism. *J. A. M. A.*, 117: 1502–1508, 1941.
5. OCHSNER, ALTON. Thrombophlebitis. *Surgery*, 6: 129, 1939.

QUESTIONS AND ANSWERS

E. PAYNE PALMER (Phoenix, Arizona): I should like to ask Dr. Allen if he has had to go into the iliac vein to ligate in any of his cases. We have had to do so in two and found the clot rather extensive when we opened up the femoral. Therefore, we made the Cameron incision, pushed the curtain in back and went back of the clot into the iliac.

DR. ALLEN: I know a doctor who has done that several times, and we have supposed that we might have to do likewise. He used that method of removal of the clot, but if you get the upper segment of the clot out, I think you need not fear fatal embolism. Any thrombus remaining above the ligature would probably stay there and not go to the lungs.

DR. PALMER: How do you know you are going to get it all out?

DR. ALLEN: I do not think it matters. You may get a small infarct as we did once in thirty-nine cases, but it is not fatal. What you are trying to avoid is enough clot going to the pulmonary artery to cause death, and this does it. Incidentally, interrupting the flow in the vein is very effectively done by the ligation of the femoral vein at this point.

Dr. Darrach asked me why an inverse incision instead of a longitudinal one. You have not much room unless you tie off small veins that are going into the muscles. There is about a centimeter between the profunda and muscle branches, which is enough if you use a transverse incision.

FREDERIC W. BANCROFT (New York City): Might I ask Dr. Allen what result he had had from blocking the sympathetic?

DR. ALLEN: We have not had as good luck with it as they have had in New Orleans. I think we have a different kind of thrombophlebitis here on the East Coast. I mean that, I have been down there and seen these cases and know they do have excellent results with this method, but our phlebitis, once it starts, is a difficult one to control. It takes six weeks to get over it; however, we have used this method very extensively and we think it has helped a great deal. It is one of the methods we would use if we were not going to ligate. Patients object to having their backs stuck too many days in succession and their phlebitis lasts too long here to make us believe we should depend upon that method alone.

KELLOGG SPEED (Chicago, Illinois): Have you used Dicoumarin at all?

DR. ALLEN: I have had absolutely no experience with it. I do not know whether that is going to be effective or not, but I hope so.

DR. SPEED: It is very effective, at least in cases I have seen.

FRASER B. GURD (Montreal, Canada): We have used Dicoumarin for the last eight or nine months, and we are of the opinion that it is useful, and as compared with heparin it costs only five cents a day.

DR. ALLEN: You say it is useful?

DR. GURD: As compared with heparin it seems to be as useful. It is given by mouth, and two or three days' administration lasts for about two weeks. The only objection that we can see is that you cannot get your clotting time back once you have compressed it, as compared with heparin.

DR. ALLEN: I do not think we have used it in this hospital as yet.

DR. GURD: I do not think it is on the market.

DR. SPEED: It is only being given out experimentally.

WILLIAM L. ESTES, JR. (Bethlehem, Pa.): How long have you kept the patients inactive after ligation of the femoral vein?

DR. ALLEN: Everything else considered, forty-eight hours is enough, unless there are other reasons for a longer period. If the patient is febrile still from his infarction, one should naturally keep him quiet longer, but forty-eight hours is sufficient in the case of the ordinary patient. For instance, the one upon whom I think I did the unnecessary ligation, I got out of bed the next day.

WILLIAM DARRACH (New York, N. Y.): How much do they swell up afterward?

DR. ALLEN: Very little if you ligate them below the profunda.

G. GAVIN MILLER (Montreal, Canada): Dr. Allen said he had had no deaths following ligation. I should like to report one. We had a patient with

an infarct; we ligated, and a few days later he died of embolism. We also had a death following ligation within six days.

Dr. Allen: Were you able to get an autopsy on the patient that died? Do you know for certain that the fatal part of the embolus came from the side you ligated?

Dr. Miller: No.

Dr. Allen: We have been pretty lucky along that line, because I am perfectly certain that patients are going to die from the side that we have not ligated. This fear has made us more willing to do bilateral ligations.

H. Gurth Pretty (Montreal, Canada): Do you find any increase in the number of these cases as the result of using cyclopropane?

Dr. Allen: Cyclopropane has not been used in this hospital since the fatal explosion that occurred in Boston. The trustees did the same thing about ethylene. We had three or four minor explosions with that and they said, "You cannot have it any more," just about the time we had become fond of it; and the same thing happened with cyclopropane. We have not used any for a number of years. I had not realized it would increase this condition.

Dr. Pretty: In looking up a great many cases of pulmonary emboli, we found that the great majority of cases in various hospitals occurred following spinal anesthesia; next in order were those following cyclopropane, and next in order those following open ether, on account of the slowing of the blood stream postoperatively.

Dr. Allen: I cannot quote you the exact figures, but when Faxon looked over our fatal cases of emboli prior to the beginning of this recent work, he sorted them out as to the kind of anesthetic agent that had been used and came to the conclusion that here, at least, we could not see that it made any difference. The greatest percentage from any department came from the urological service, probably because they were dealing with a much older age group.

FRACTURES OF THE METACARPALS EXCLUSIVE OF THE THUMB*

A NEW METHOD OF TREATMENT

RICHEY L. WAUGH, M.D.   AND   G. P. FERRAZZANO, M.D.

Chief of Surgical Service, U. S. Marine
Hospital

Assistant Surgeon, U. S. Marine
Hospital

BOSTON, MASSACHUSETTS

FRACTURES of the metacarpals, although commonly regarded as trivial, are actually serious injuries frequently resulting in crippling deformities due to faulty union and contractures of the finger joints. This is largely a result of the difficulty in reducing the fracture and maintaining the proper position. Other factors are the lack of interest and the less consideration generally given to the early treatment and immobilization of these fractures than that given to fractures of the larger long bones.

It is admitted that simple splinting is quite satisfactory in fractures of the metacarpals without displacement. On the other hand we are convinced that the deformities (Fig. 1) and disabilities which follow metacarpal fractures with displacement are due to the failure to properly reduce the fractures by the usual methods and the inability or failure to maintain the reposition (restoration of bone length and prominence of the knuckle). The latter is influenced no doubt by the endeavor to prevent loss of joint function by too early mobilization of the finger joints.

Prior to 1932, practically all metacarpal fractures were treated by simple bandaging over a roller bandage with little or no attempt made to correct the displacement.

In 1932, McNealy and Lichtenstein[1] pointed out the unsoundness of the roller bandage method of treatment and called attention to the mechanics involved in these fractures and advocated treatment with a straight dorsal splint with the hand in extension. Since then the trend has been to immobilize these fractures in extension, frequently using traction, with either a straight dorsal or a banjo splint. This form of treatment has been recommended by Scudder,[2] Key and Conwell,[3] Magnuson,[4] Christopher[5] and others. In this connection Cotton[6] stated "there is no longer any excuse for treatment with the hand flexed either on the once much used roller bandage or the more

modern aluminum substitute." Owen[7] also condemned the use of "a roller bandage as a panacea for fracture of metacarpals."

Although the so-called extension treatment with or without the

FRACTURES OF METACARPALS

Normal

Metacarpal

Middle shaft

Oblique Fracture

FIG. 1.

Distal End (Knuckle)

Transverse Fracture

Middle Shaft

Transverse Fracture

FRACTURES OF METACARPALS

Traction

Trapeze

Counter-
pressure

M. Interosseus

Pressure

Countertraction
(weight of forearm)

FIG. 2.

MOTIONS OF METACARPOPHALANGEAL JOINT

Flexion

Taut Collateral
Ligaments

Extension

Motion
All Directions

Flexion

No Lateral Motion
Metacarpal Rotates
With Proximal Phalanx

FIG. 3.

Extension (Tenses interosseous muscles)   Angulation Maintained
          (Relaxes collateral ligaments)   No Control Distal Fragment

Flexion   (Relaxes interosseous muscles)   Direct Control Distal Fragment
          (Tenses collateral ligaments)   Reduction by Distraction and
                                           Compression Forces

FIGS. 1–3.

use of traction indicated progress in the treatment of fractures of the hand, joint dysfunction due to capsular contractures of the fingers

frequently occurred after this form of treatment. Koch[8] states that fixation of the finger joints are prone to occur with the metacarpophalangeal joints immobilized in extension or with the proximal
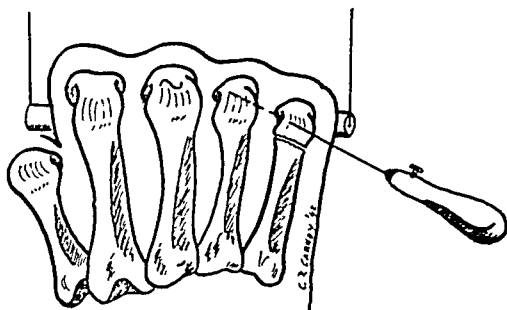
FIG. 4.

interphalangeal joints immobilized in flexion. To prevent these contractures Koch advocates immobilization of the hand in the optimum position for function, i.e., position of grasp.

Further progress was made when Jahss,[9] in 1938, reported a new method of treatment for the reduction of these fractures based on the

Cork Capped Over End
of Projecting Wire

Plaster Dressing with Hand
in Position of Grasp

Plaster Dressing Applied
Incorporating Cork over Pin

Plaster Dressing with Hand
in Position of Extension
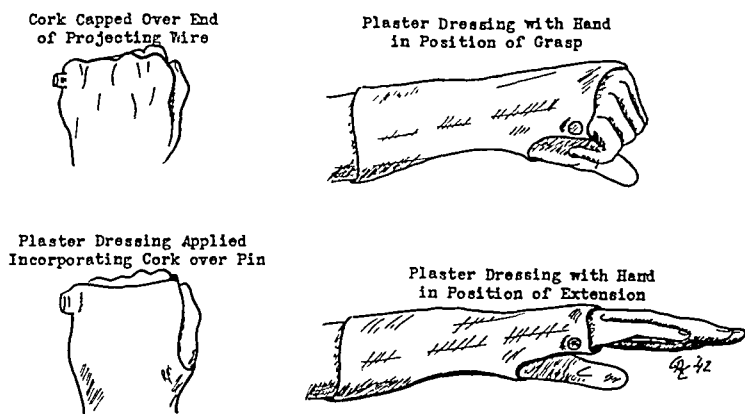
FIG. 5.

correction of the anatomical deformity with the metacarpophalangeal and interphalangeal joints in flexion, and also with immobilization of the affected fingers in flexion.

As indicated by Jahss we have found that the reduction of the deformity is facilitated with the metacarpophalangeal and proximal interphalangeal joints of the involved finger held at 90 degrees. Unfortunately, however, plaster immobilization of the fingers in flexion usually results in limitation of extension power of the fingers.

Such contractures frequently require intensive physiotherapy for a considerable period after the removal of the cast. In an endeavor to correct the anatomic deformity and at the same time to prevent the restriction of finger motions which follow the immobilization of the finger joints we present a new method of treatment for these fractures.

TABLE I

AGE RANGE AND METACARPAL INCIDENCE

Ages: 15 to 47
Hand: Left 5; Right 17
Metacarpal: Single 20; Multiple 2
 (Both multiple fractures were of 4th and 5th)
 5th Metacarpal—15 3rd Metacarpal—4
 4th " — 3 2nd " —2

TABLE II

FRACTURES OF METACARPALS ACCORDING TO CLASS OF WORKER AND MODE OF INJURY

Class of Worker	Mode of Injury				Total
	Fight	Falling or Moving Objects	Fall	Struck or Jammed Hand	
Industrial	0	3	3	6	12
Seamen	3	1	1	2	7
CCC	1	0	1	0	2
Schoolboy	0	1	0	0	1
Totals	4	5	5	8	22

TABLE III

METHODS AND RESULTS OF TREATMENT

Methods of Treatment:
 Closed treatment—16 Open treatment—6
Indications for the Open Operations:
 Delayed union—1 Compound fracture —1
 Faulty union —3 Fracture-dislocation—1
Results (based on function and absence of deformity):
 Excellent 14 Satisfactory 7 Poor 1

The method of treatment has been used in twenty-two cases of fractures of the four inner metacarpals, including both shaft and so-called "knuckle" fractures. The method is believed to be superior to the older methods of treatment.

The basis of treatment, as indicated above, is the early correction of the primary anatomic deformity of the fracture and the rigid fixation of the fragments, with the hand in the position of grasp (opti-

A

B

FIG. 6. Roentgenogram showing fracture of distal end of fifth metacarpal, A, before reduction and B, after reduction with fixation by pin and plaster.

FIG. 7. Roentgenogram showing fracture of middle shaft of fifth metacarpal; A, before treatment; and B, after treatment.

A   B   C
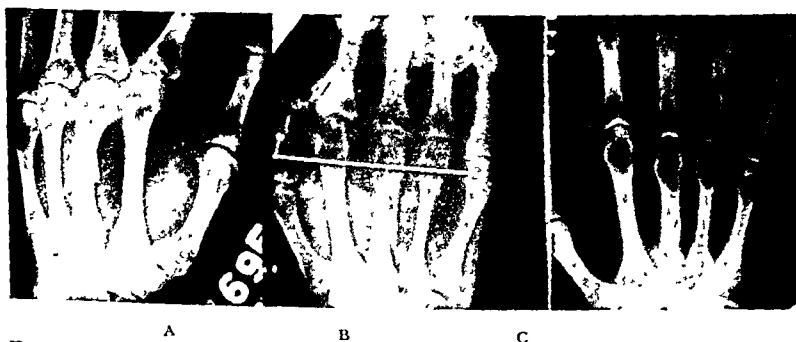
FIG. 8. Roentgenogram of fracture followed by delayed union, third metacarpal; A, ten weeks after injury; B, after open operation (intramedullary bone graft and fixation by pin and plaster); and C, one year after open operation (good anatomical and functional result).
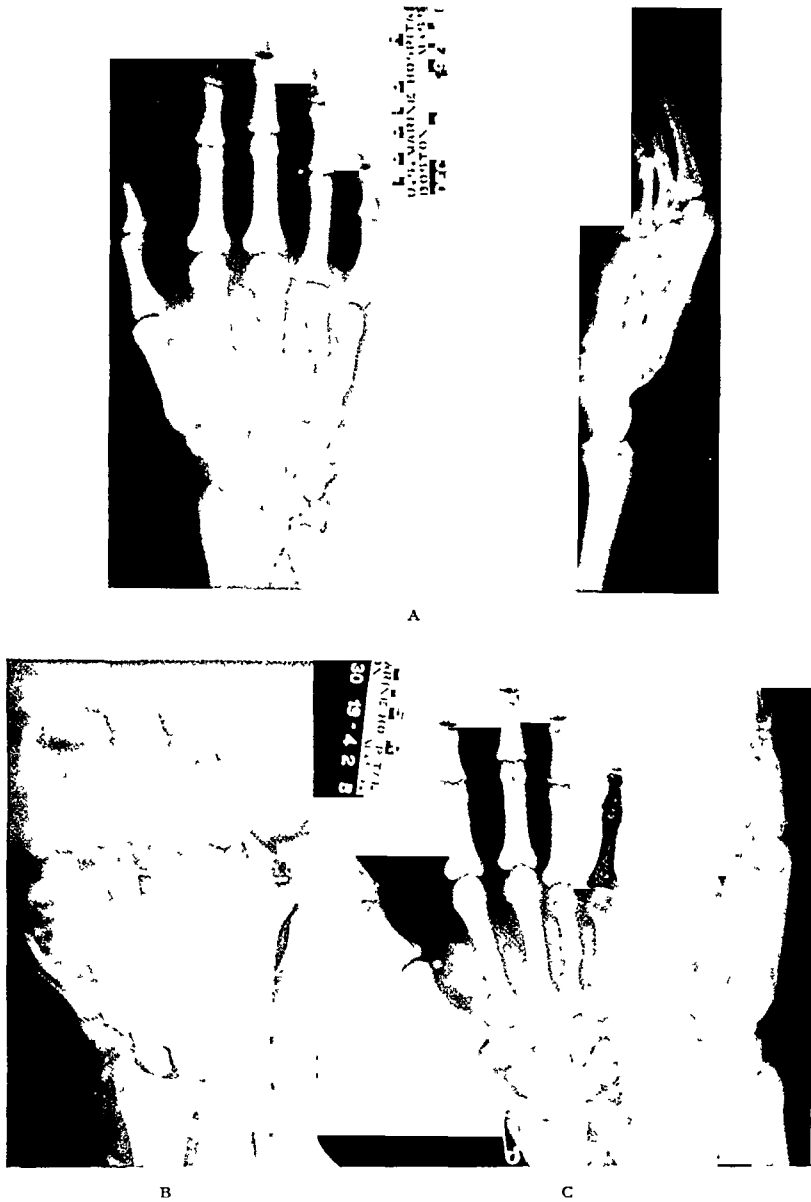
Fig. 9. Roentgenogram showing fractures of fourth and fifth metacarpals; A, before reduction; B, after reduction and fixation by pin and plaster; and C. six weeks after reduction.

mum function), but with all metacarpophalangeal and interphalangeal joints free during the period of immobilization.

The reduction of the fracture is accomplished by closed methods. The forearm is suspended vertically with the hand grasping a small trapeze. (Fig. 2.) The phalanges flexed over the small cylinder of the trapeze produces the traction and the weight of the forearm and arm the countertraction forces. The flexed proximal phalanx relaxes the interosseous muscles and tightens the collateral ligaments (Fig. 3) and synovia (capsule) of the metacarpophalangeal joint, thus allowing positive control of the distal fragment of the fractured metacarpal. The actual reduction of the fracture is effected by compression by means of the surgeon's left index finger and thumb. (Fig. 2.) The pressure is applied over the flexed proximal interphalangeal joint, and the counterpressure over the dorsum of the hand (the site of the dorsally bowed metacarpal fragments).

With the fracture reduced by the distraction and compression forces, rigid fixation is secured by means of a short Kirschner or Mathews wire about 2½ inches in length inserted transversely as a "pinion" through the distal ends (heads) of the fractured and adjacent one or two metacarpals. (Fig. 4.) Novocain solution is used as a local anesthetic necessary at the site of the insertion of the wire and between the heads of the fractured and adjacent metacarpals.

Finally, a very light plaster of Paris or starch bandage, incorporating a small cork capped over the ½ inch of wire protruding through the skin (the site of insertion) is applied to the hand and distal half of the forearm. The fingers are not encased in the plaster or starch dressing but are free permitting motion of the metacarpophalangeal and interphalangeal joints. (Fig. 5.) The continued free motion prevents capsular contractures of the finger joints so frequent after the usual methods of immobilization, particularly after immobilization in extension. The immobilization (wire fixation with plaster or starch bandage dressing) is continued for at least three weeks.

Usually after a week of physical therapy in the form of whirlpool and increasing active use of the hand, the patient can return to his usual work.

SUMMARY*

1. The seriousness of metacarpal fractures, commonly regarded as trivial injuries but frequently resulting in crippling deformities is

* Since the presentation of this paper, the authors have treated ten additional pa-

emphasized. These disabilities far in excess of the apparent importance of the fracture are due, to a considerable extent, to the lack of interest and the less consideration generally given to the early treatment and immobilization of these fractures than that given to fractures of the larger bones.

2. The difficulties of treatment with special reference to past progress made in the treatment of these fractures are briefly reviewed and discussed.

3. A new method of treatment used in twenty-two cases of fractures of the four inner metacarpals including both shaft and the so-called knuckle fractures is presented. This method is believed to be superior to the older methods of treatment. The basis of treatment is the early correction of the primary anatomic deformity and the rigid fixation of the fragments, with the hand in the position of grasp (optimum function) but with all metacarpophalangeal and interphalangeal joints free during the period of immobilization.

4. The results of treatment are shown in the accompanying tables and representative roentgenograms.

REFERENCES

1. McNealy, R. W. and Lichtenstein, M. E. Fractures of the metacarpals and phalanges. *Surg., Gynec. & Obst.*, 55: 758–761, 1932.
2. Scudder, C. L. The Treatment of Fractures, 11th ed., pp. 763–772. Philadelphia and London, 1938. W. B. Saunders Company.
3. Key, J. A. and Conwell, H. E. Management of Fractures, Dislocations and Sprains. Pp. 699–703. St. Louis, 1934. C. V. Mosby Company.
4. Magnuson, Paul B. Fractures of metacarpals and phalanges. *J. A. M. A.*, 91: 1339–1340, 1928.
5. Christopher, Frederick. Minor Surgery, 4th ed., chap. 18. Philadelphia and London, 1940. W. B. Saunders Company.
6. Cotton, F. J. Ten years of progress in the treatment of fractures. *Illinois M. J.*, pp. 317–329, October, 1934.
7. Owen, H. R. Fractures of bones of hand. *Surg., Gynec. & Obst.*, 66: 500–505, 1938.
8. Koch, Sumner L. Disabilities of hand resulting from loss of joint function. *J. A. M. A.*, 104: 30–35, 1935.
9. Jahss, S. A. Fractures of metacarpals; a new method of reduction and immobilization. *J. Bone & Joint Surg.*, 20: 178–186, 1938.

tients. In these cases, the actual reduction was accomplished solely by manipulation (compression forces with the fingers in flexion) without the use of the trapeze and using intravenous pentothal-sodium for anesthesia. The results were excellent in all cases. The authors, therefore, recommend this modification as the method of choice for the reduction of these fractures but with the same wire fixation and plaster dressing as described in their original manuscript.

EXPERIENCE WITH BURNS AT THE
HOSPITAL FOR SICK CHILDREN

Alfred W. Farmer, m.d.
On Staff of Hospital for Sick Children
TORONTO, CANADA

DUE to the limited space, it was decided that rather than stimulate any debate concerning the relative merits of different forms of therapy, to tell the realities as we have found them. With minor exceptions my ideas on results will not be presented, as they would add little to the already voluminous writings on the subject. Certain events concerning burns, however, appear to have been largely overlooked or to lack emphasis in the literature. It is chiefly to these that attention will be drawn. Thus this paper deals principally with facts about cases treated as in-patients from 1913 to 1942, all of these since 1932 being looked after by the author.

Nineteen-thirteen was picked, as from this date suitable records have been kept and are available. The cases which number over 1,800 are divided into four periods. In August, 1925, tannic acid was used first, so that this represents the end of the first period. Statistics until 1928 were gathered by Dr. R. I. Harris, who was then on the hospital staff. Thus this time has been taken as the end of the second interval. The third goes to the end of 1937, when sulfonamides were first used in our cases of burns, and the last is until the end of 1941.

The table (Table 1) by which the area of surface affected was deduced is shown. This is practically the same as Berkow's table. Allowances were made for discrepancies in this due to dealing with children of different ages. Until 1928, Dr. Harris made rough measurements of the areas and used no specific chart.

The second table shows the mortality statistics for the period 1913 to 1925. The causes for death have been divided into shock, toxemia and sepsis. These terms as used here must be defined. Listed under shock are those who died within ten hours of the accident, provided they did not show the signs ascribed to the next stage. The shock as herein described is what is called primary shock. It is my opinion that the term primary shock as applied to burns might well be dropped, and this column be added to the next. The shortest time in this whole series between accident and death was two hours, which is plenty for the development of the general reaction to a severe burn.

The term, "toxemia," as used here is referred to more commonly as secondary shock, or occasionally secondary shock and toxemia. Because of my belief that toxins cause the "oligemic anoxia" called

TABLE 1

PERCENTAGE OF TOTAL BODY SURFACE OF COMPONENT PARTS
AS USED AT THE HOSPITAL FOR SICK CHILDREN

HEAD	6%					
NECK	2%					
BODY	38%	FRONT 12	BACK 12	ONE SIDE	7	(X2)
UPPER LIMB	16%	ONE ARM 3½	ONE FOREARM 2½	ONE HAND	2	(X2)
LEGS	30%	ONE THIGH 11	ONE LEG 6	ONE FOOT	2	(X2)

secondary shock, as well as the other findings in this stage, all patients dying with these features have been listed under the one heading. This group of signs and symptoms are easily noted in burns of moderate or severe degree. They include an elevated temperature. This is usually about 101° to 102°F. and varies somewhat depending upon a number of factors, such as the extent of burn, the size of patient, etc. There is a fast pulse rate out of proportion to the temperature. Next, there is drowsiness, which may go on to coma and death. This drowsiness is occasionally disturbed by periods of irritability. Often there are localized twitchings and sometimes generalized convulsions. Vomiting commences early in severe burns. It may become continuous and in appearance is like coffee grounds. There is bright red blood occasionally. The limbs become moist and cold. One can blanch a grayish blue area on a limb and the return of color is very slow. The blood pressure falls eventually. The urine is of small volume and has a high specific gravity. Albumin may appear in severe cases as well as hyaline and granular casts. Acetone may be present.

Death while the above mentioned clinical features are manifest, has been listed under toxemia in this classification. The earliest was about four hours after the accident, although most occurred in three to four days, while a small number of cases were as late or later than ten days. If the blood is examined, it is found that changes have happened before the clinical findings are noted, great emphasis being placed in recent years on the hemoconcentration. While these changes occur before the clinical signs and symptoms, they may be largely or completely corrected before the patient dies. Under the column of sepsis are listed the cases who had a swinging type of fever and discharge from the burn area. The blood culture may or may not have been positive.

During the period 1913 to 1924 (Table II) the average mortality rate was 35.2 per cent. The shock and toxemia columns take in most of the deaths. The yearly admissions rose during this time to nearly

TABLE II

YEAR	TOTAL BURNS	DEATHS	SHOCK	TOXAEMIA	SEPSIS
1913	11	6(55%)		6	
1914	18	7(39%)	2	4	1
1915	22	8(36.4%)	5	3	
1916	37	14(37.8%)	2	8	4
1917	47	18(38.3%)	7	9	2
1918	29	10(34.5%)	6	1	3
1919	35	11(31.4%)	2	8	1
1920	38	13(34.2%)	3	7	3
1921	35	13(37%)	2	7	4
1922	46	13(28.3%)	3	8	2
1923	48	10(20.8%)	1	7	2
1924	46	14(30.4%)	5	5	4

fifty. The local treatment varied between vaseline, picric acid, ambrine and sodium bicarbonate. The general therapy included infusions of saline, calcium chloride and calcium lactate. The first transfusion was performed in 1919. The first exsanguination transfusion was in 1920. The average survival time excluding deaths after ten days was 1.7 days. A large number died in the first twenty-four hours.

TABLE III

YEAR	TOTAL BURNS	DEATHS	SHOCK	TOXAEMIA	SEPSIS
1925	60	10(16.6%)	1	5	4
1926	52	9(17.3%)	2	2	5
1927	68	10(14.7%)	1	4	5
1928	50	8(16%)	1	4	3

The next table (Table III) takes the same statistics from 1925 to 1928 inclusive. The average number of admissions increased a little. The mortality rate dropped suddenly to approximately one-half of those previously recorded, now being 16.1 per cent. The first column claimed fewer lives. The percentage of deaths due to sepsis when compared to the addition of the other two columns rose. In this period local treatment was tannic acid. General treatment included whole blood transfusions, exsanguination transfusions and intravenous electrolytes. The average survival time (excluding death after ten days) was 3.4 days, which represents a considerable improvement over that of the previous period.

Table IV takes the data until 1937. The average number of admissions increased to eighty-five. The mortality rate dropped still further to 11.8 per cent. Toxemia claimed most lives. Sepsis still

TABLE IV

YEAR	TOTAL BURNS	DEATHS	SHOCK	TOXAEMIA	SEPSIS	OTHER CAUSES
1929	72	7(9.7%)	1	2	2	2
1930	87	14(16.1%)	2	8	3	1
1931	84	8(9.5%)	1	7	0	0
1932	90	16(17.7%)	0	9	2	5
1933	116	12(10.3%)	0	8	4	0
1934	62	7(11.3%)	0	5	1	1
1935	106	6(5.7%)	1	4	1	0
1936	80	11(13.8%)	0	4	4	3
1937	85	10(11.8%)	1	9	0	0

played a part but this was less serious. A new column was introduced, because in a children's hospital due to intercurrent infections, it was impossible to ascribe all the deaths to the foregoing three columns.

TABLE V

BURNS TREATED WITH FERROUS OR FERRIC CHLORIDE

NUMBER		EXTENT
56090	621 B	Less than 5%
60058	672 A	5-10%
67076	623 A	5%
66986	673 B	Less than 5%
71938	624 A	5-10%
81884	651 B	5-10%
82110	652 B	5%
82223	655 A	5%
83364	661 A	30%
83579	676 B	5-10%
83660	665 A	5%
83678	666 A	10%
83791	666 B	5%
83793	668 A	5-7%
83908	668 B	5%
83934	669 A	5-10%
84055	669 B	5%
84088	670 A	5%
84397	678 A	5-10%
84611	679 A	Less than 5%
84640	679 B	15%
84720	680 A	5-10%
84723	680 B	35%-----Death
84851	681 A	5-10%
84922	681 B	Less than 5%

One death (35%) burn - considerable trouble due to infection -
toxaemia noted in some cases but extent of burn not great
except in one case of sunburn.

The local treatment still remained mainly tannic acid. Whenever any new idea seemed to merit, it was tried on a selected number of cases. For example Table V shows the twenty-five patients on whom ferric and ferrous chloride solutions were used. The general treatment still consisted of blood transfusions and intravenous electrolytes in the early stages. Exsanguination transfusions were discontinued, the last being performed in 1930. The survival time (excluding deaths after

ten days) was 3.8 days, which represents a slight improvement over the preceding period.

Table VI shows the period from 1938 to 1941 inclusive. The

TABLE VI

YEAR	TOTAL BURNS	DEATHS	SHOCK	TOXAEMIA	SEPSIS	OTHER CAUSES
1938	100	4(4.0%)	0	2	1	1
1939	58	1(1.7%)	0	1	0	0
1940	108	1(.9%)	0	1	0	0
1941	107	5(4.7%)	0	5	0	0

TABLE VII

MORTALITY RATES

1913 - 1924	35.2%
1925 - 1928	16.1%
1929 - 1937	11.8%
1938 - 1941	2.9%

TABLE VIII

AVERAGE SURVIVAL TIME

EXCLUDING DEATH AFTER 10 DAYS

1913 to 1924 inclusive	1.7 days
1925 to 1928 "	3.4 days
1929 to 1937 "	3.8 days
1938 to 1942 "	5.0 days

average number of admissions except for a small year in 1939 was over 100. The mortality rate for this period was 2.9 per cent, which is a marked drop. The deaths are practically all due to toxemia, there being none from sepsis in the last three years. The average survival time (there were no deaths after ten days) was five days. The early local treatment in this period was chiefly tannic acid. Sulfonamides were used locally and generally in the later stages. General treatment in the early stages consisted of blood transfusions, normal and concentrated serum and intravenous electrolytes. Cortin has been used since 1939 in severe cases. Table VII compares the mortality rates in the periods described and Table VIII gives the average survival time (deaths after ten days excluded). While the ten-day exclusion date was chosen because of the prevalence of contamination or infection in deep burns after the lapse of such a period, we have had patients die after this time with what we have listed as toxemia, as there was

no clinical evidence of infection. The toxemia of infection, when added to the severe illness of the early stage, is more likely to cause death, when either factor alone may not produce such an end.

TABLE IX

BURNS

MORTALITY ARRANGED ACCORDING TO
THE EXTENT OF THE BURN 1913-1928

	NUMBER	DEATHS	MORTALITY
10% or less of total skin surface	391	30	7.7%
10 to 20%	134	52	38.8%
20 to 30%	34	23	67.6%
30 to 40%	35	24	68.6%
40 to 50%	16	16	100.%
50 to 60%	11	11	100.%
60 to 70%	7	7	100.%
70 to 80%	11	11	100.%
80 to 90%	3	3	100.%
	642	177	27.5%

TABLE X

BURNS

MORTALITY ARRANGED ACCORDING TO THE
EXTENT OF THE BURN (1929-1937)

	NUMBER	DEATHS	MORTALITY
5% or less	473	11	2.3%
5-10%	174	8	4.6%
10-20%	65	20	30.8%
20-30%	39	26	66.6%
30-40%	11	7	63.7%
40-50%	8	7	87.5%
50-60%	4	4	100.%
60-70%	· 4	4	100.%
70-80%	1	1	100.%
80-90%	2	2	100.%
90-100%	1	1	100.%
	782	91	11.6%

The cases in the four periods as previously outlined were then investigated as to the mortality rates depending on the extent of the burn. The first two periods are put together in Table IX. It is to be noted that no cases with over 40 per cent of the surface area affected lived. The mortality rate rose very rapidly after 10 per cent and approximately one in three patients between the 10 and 20 per cent areas died.

Table x takes the same statistics until the end of 1937. Here the cases were further broken up into area of 5 per cent or less, and areas between 5 to 10 per cent. It is seen that there is practically no change

TABLE XI

BURNS

MORTALITY ARRANGED ACCORDING TO THE EXTENT
OF THE BURN (1938-1941)

	NUMBER	DEATHS	MORTALITY
5% or less	247	1	0.4%
5-10%	81	1	1.2%
10-20%	32	1	3.3%
20-30%	6	2	33.3%
30-40%	3	2	66.6%
40-50%	2	2	100.%
50-60%	2	2	100.%
60-70%	0	0	100.%
70-80%	0	0	100.%
80-90%	0	0	100.%
90-100%	0	0	100.%
	373	11	2.9%

between these figures and those of the preceding table, which mainly represented forms of therapy other than tannic acid. It must be remembered, however, that the former table concerned the cases until 1928. The figures for surface area were obtained by Dr. R. I. Harris, and it is my opinion from review of the charts of these cases that there was some error of estimation on the high side. Thus a truer comparison between this and the preceding table is that of total mortality rates, which it is seen have fallen greatly. In the statistics since 1928 erythema was not included in the estimation of the surface affected.

Table xi takes the same statistics until the end of 1941. While the total number is approximately one-half that of the two preceding tables, the mortality rates have fallen markedly in the 10 to 20 per cent groups, and the 20 to 30 per cent groups, where it is the rule rather than the exception now to have the patient live.

Table xii analyzes the reasons for death in the burns of 5 per cent or less from 1929 to 1942. Sepsis and intercurrent infection have been the main causes. Toxemia has been listed in two cases in which there is a possibility for difference of opinion. This explains the query after each case. The main thought to be derived from the foregoing is that burns under 5 per cent in area are not serious from the standpoint of secondary shock or toxemia.

Many of the mortalities were followed by postmortem examination. The findings were remarkably uniform depending upon the period between accident and death. From the examination of reports

TABLE XII

BURNS

PERCENTAGE INCIDENCE OF BURNS OF 5 % OR LESS

IN EXTENT BY YEARS

			MORTALITY % FOR YEAR	MORTALITY FOR 5% BURNS
1929	55	76.3%	9.7%	3 (1 sepsis) (2 intercurrent)
1930	55	63.2%	16.1%	3 (1 toxaemia?) (1 sepsis) (1 intercurrent)
1931	50	59.5%	9.5%	0
1932	40	44.4%	17.7%	1 (intercurrent)
1933	66	56.9%	10.3%	1 (sepsis)
1934	39	62.9%	11.3%	1 (sepsis)
1935	70	66 %	5.7%	0
1936	54	67.5%	13.8%	2 (2 intercurrent)
1937	43	50.5%	11.8%	0
1938	68	68.0%	4.0%	1 (toxaemia ?)
1939	31	53.5%	1.7%	0
1940	80	74.1%	0.8%	0
1941	68	63.5%	4.7%	0

TABLE XIII

CORTIN 14 CASES

HISTORY NUMBER	TIME OF DEATH(DAYS)	EXTENT OF BURN	AGE IN YEARS
97951	4	46%	8
110888	2	55%	3
114153		Less than 10%	1
115710	7	20%	3
116368	2	40%	1
10625	-	30%	6
115751	-	10%	3
95280	-	25%	11
95740	4	50%	2
118082	3	40%	9 mos.
120152	-	10%	4
120554	3	30%	9
120663	5	25%	2
121997	-	25%	3

14 cases---8 deaths---average extent 38.2---average survival

time 3.6 days

6 survivals---average extent 18.3%

of fifty-six autopsies between 1920 and 1942, the following items were found:

Following death in the first twenty-four hours (twelve cases) there was mainly congestion of viscera, with petechial hemorrhages in the organs and connective tissues. This was most noticeable in liver, lungs, spleen, lymph glands, kidneys and adrenals, and in the

subpleural and subpericardial connective tissues. The head was not often included in the examination, but when it was, the same condition obtained here. Congestion in the gastrointestinal tract was not prominent at this time. It seemed to begin in the first part of the duodenum with more widespread involvement later. Rarely there were beginning fatty changes in the liver, and in one case there was some degeneration in the tubules of the kidneys.

Following death in the forty-eight-hour period (nine cases) the preceding changes were more accentuated. The congestion was more marked, petechiae were widespread and perhaps larger. Edema made its appearance. The liver changes were more advanced, with central liver necrosis. Two cases showed nephrosis. There were cases of congestion and erosion (not deep ulceration) of the mucous membrane in areas in the esophagus, stomach and duodenum. The lymph glands were edematous, large, and congested, and showed petechial hemorrhages. This included the regional as well as the mesenteric and mediastinal glands. Quite often the term, acute mesenteric lymphadenitis, was used in the description. In one microscopic report it was noted that there was an increase in reticulo-endothelial cells and some of these showed phagocytosis for broken down red blood cells.

In death in three days (seven cases) liver damage was the most striking feature. There was extreme fatty degeneration. Degenerative changes in the kidney epithelium were more advanced. The lungs were congested and edema was greater. In death in four days (seven cases) the liver alteration progressed to actual necrosis with loss of large portions of the central vein area, collapse of the sinusoids and hemorrhage. The few remaining cells contained fat vacuoles and appeared degenerating also. All of the cases at this time showed nephrosis. The hepatic, and renal changes were the outstanding features. The other fairly constant findings were pulmonary congestion and occasional small hemorrhages in many organs.

In death in five to seven days (six cases) there were no additional features. There was seen a diminution in the intensity of the previous changes. Five of the six cases autopsied at this time showed early bronchopneumonia. This is an important factor for those treating burns to remember. The adrenals were still congested and there was one duodenal ulcer in this group.

In death in eight to ten days (four cases) very interesting changes were found. There was evidence that healing was taking place in the liver. The liver cell débris was gone. There was still some fat in the

periportal region, but in the center of the lobule regenerating liver cells were present. There was some hemosiderin deposition in the Kupffer's cells. The kidneys, however, showed more advanced changes than seen before. The convoluted tubules were dilated, epithelium was degenerating, and granular casts appeared in them and in the collecting tubules.

In death in eleven days to twelve weeks (eleven cases) the picture is not so clear. Infection with septicemia had been added in eight out of eleven. The organisms were Staphylococcus aureus in two, haemolytic streptococcus in three, and Bacillus pyocaneus in three. The kidney changes became insignificant.

While the facts as given are in general true, it is only fair to state that there is much variation in the individual picture. Thus, a two-year old child dying of a 20 per cent scald in four days showed extensive liver necrosis, while a five-year old with a 60 per cent fire burn, dying after the same interval, showed only moderate and early central necrosis.

The question of exsanguination transfusion will be briefly discussed. The experience of our hospital in this regard was never published. It was begun in 1920 by Dr. Bruce Robertson, who had a particular interest in burns, and discontinued in 1930. There were eighty-five transfusions performed during this time for burns and still more for other conditions. It consisted in the withdrawal of 50 cc. of blood per pound of body weight and the replacement with a slightly greater amount from a number of donors. The withdrawal was started first, and after an interval the two acts proceeded together. Many cases appeared to be improved following the operation. It was because of this that the procedure was continued for ten years. As much as 1,000 cc. of blood were given to a one-year old child which was later repeated. The operation was difficult and lengthy and was attended with a certain mortality rate directly attributable to it. Thus it was finally discontinued.

Certain other facts concerning the cases are worth mentioning. Many years ago there was a 10 per cent incidence of surgical scarlet fever in the patients with burns. Since the introduction of a routine dose of scarlet fever antitoxin, there have been two cases only in considerably over 1,000 burns. One of these received the prophylactic dose. The other was an infant to whom it was not given as it was planned to discontinue its use after the inception of the sulfonamides.

Five cases only developed visible jaundice. Two of these recovered. There were only two cases of gross hematuria. Many cases

vomited bright red blood. This did not mean necessarily a deep duodenal ulcer. Postmortem examination usually showed a superficial erosion of the lower part of the esophagus, stomach or duo-

FIG. 1. S. G., age six years; suffered 60 per cent burns. The patient died in fifteen days from sepsis.

denum. About one in ten admitted cases since 1928 required skin replacement. While grafting has been performed in less than one month from the time of the accident, following tanning, this is the exception, and six weeks is nearer an average.

Three cases of gangrene of the fingers have been seen. None of these were treated by tannic acid. It is thought that the very great reduction in peripheral circulation together with the trauma is probably the main cause of this complication and not the treatment. Recently in the literature the firm tannic acid eschar has been condemned in this regard.

While it is obvious from the statistics that all patients with burns of large surface area have died, attention is drawn to the fact that many of the patients with the largest fire burns, which are presumably deeper on an average than scalds, lived for considerable lengths of time. One such example, of which there were many, is shown in Figure 1.

Concerning the value of cortin, we are unable to make any definite statement. It has been used since 1939 in fourteen cases. It has been reserved because of expense for those cases in which it was surmised that death might ordinarily be the end result, and for those in which the clinical picture was very serious even though the extent of the burn was not so great. It was started frequently after a number

of days and only when the patient became suddenly seriously ill. In such a case there was always a rapid improvement, which, however, might last for a few hours only. This improvement was judged by clinical observations, there being a return from unconsciousness often, a raised blood pressure, from one which could not be recorded, etc. Cortin* is supposed to be free from pressor or depressor substances. In the cases of burn it has been used in large doses. For example, a three-year old boy with a 25 per cent fire burn was started twelve hours after the accident on 25 cc. intravenously and continued with 5 cc. every two hours for four days. This means a total of 80 cc. in the first twenty-four hours, and 60 cc. a day thereafter. He did not show the expected degree of hemoconcentration, nor did he show the usual clinical signs in great severity. However, as stated previously, while the impression is that cortin is of value, no definite inference can yet be drawn from our cases.

In conclusion, interesting features from over 1,800 acute burns of which approximately 1,000 were under my care have been discussed. In the last three decades there has been a progressive rapid fall in mortality rates, both when based on numbers of hospital admissions and on area burned. The first great reduction (35.2 to 16.1) was coincident with the use of tannic acid. Following this there was a gradual reduction until the final period when there was again a sudden rapid reduction (11.8 to 2.9 per cent). This was coincident with more intensive early general treatment, but other factors may also have played a part. It is thus emphasized that intensive early general therapy must not be neglected in large burns; also while there is as yet no ideal local application for all burns, the changing of a large wet wound, often painful, into a dry comfortable surface (with such a substance as tannic acid) remains an excellent form of therapy. Infection has been a negligible factor in mortality in recent years and a minor cause for morbidity. It is not believed that the number of cases proceeding to grafting after coagulation therapy is greater than after other types of treatment.

* The cortin used is manufactured by the Connaught Laboratories, University of Toronto, and dispensed as a sterile aqueous solution, biologically standardized to contain 30 dog-units per cc.

DISCUSSION

J. HUBER WAGNER (Pittsburgh, Pa.): The thought came to me, as I saw these wonderful statistics presented of this marked drop in death rate due either to shock, shock toxemia, or what you will within five days or forty-eight hours, that we cannot attribute this all to the method of treatment but to the fact that burns are being recognized as major surgery and the patients are now being sent to hospitals, whereas in previous years many of these were treated and died at home. The minor burns that were once treated at home are now being treated in a hospital; therefore we know that as the number goes up, the classification is more widespread and it gives a chance for these statistics to be brought down. I am not belittling the paper in any way or the method of treatment, but the fact remains that that is a possibility.

The fact still remains that burns are major surgery and if treated as major surgery such as we know with these modern aids, transfusions, plasma and proper local treatment, our results will be better.

I should like to have heard the statistics of the number of days in the hospital, but that was not available. But we do know that in many of our cases of burns, first and second classification, the stay in the hospital is very limited, whereas with a third or a little beyond a third, a fourth, if you will, if the burned area does not extend over 40 per cent, with our modern methods we have an excellent chance of saving the child's life, and with later skin grafting, of bringing him around to a normal, healthy individual again.

I am quite in accord with the modern trend as I believe that with the use of tannic acid, there is quite a possibility of tanning some tissue that might, if treated by some milder procedure, still live and be viable; whereas if it is on the border, tannic acid takes away what little live tissue might be left and may possibly cause a little deeper burn in itself.

I think the method of compression treatment that has recently been advocated by Siler is quite in accord with what has been done in our clinic since 1915 and 1916. We still believe that the paraffin wax is an excellent method of treatment. It allays pain immediately, and when properly applied, after the first dressing causes no discomfort whatever.

The eschar method, we know, is certainly of minimum value when compared with tannic acid. The thought that has been brought out by Blair, of compression intelligently applied, with something definite in mind, has, I think, been an excellent contribution. We believe that this modern treatment of vaseline plus the sponge dressings is in a sense what we have been doing with wax for a number of years, and it is my opinion now that it is our tendency, rather than to treat these patients every second day, to carry our wax dressings as long as six or eight days with good effects.

Dr. Farmer's paper is an excellent presentation. I think the work done at Toronto Sick Children's Hospital is as good if not better than that done at

any place I have known. We do not keep statistics. Ours is not a teaching institution, so what we have are mostly impressions rather than the actual facts; but I believe, by and large, that we save as many children as has been shown here, and I know that we are getting many more burns to treat that are less severe than has been the case in the past.

E. PAYNE PALMER (Phoenix, Ariz.): For the past two years we have been carrying on experimental work in shock, and we believe that the emergency treatment of shock in burns is as important as in fractures. These patients should be treated at the site of the burn. First relieve the shock and then treat the burn. Many of these patients die of shock on the way to the hospital or shortly after they arrive in the hospital. Therefore, we must have a remedy which we can administer at the place of the accident.

Using a number of chemicals and drugs, we have found that we have had only one that has given us satisfactory results. These experiments which we have carried out show that there has been a depression of the muscle tone, a depression of the venous system and a slowing down of the blood current, and the only remedy that we have been able to give that could stimulate muscle tone, lowered venous tone and bring about a return of the venous flow has been coramine. I will not give you the chemical name because it is rather complicated. We found that it is necessary to give this in large doses, both to children and to adults, depending upon the age of the child. The adult that is unconscious should get it intravenously and not intramuscularly, because if you give it intramuscularly or subcutaneously, you will get no effect that will relieve the shock.

The conscious patient should be given 5 cc., and repeated 10 or 5 cc. as necessary in the child, in proportion to its age. Shortly after the administration, the patient takes a deep breath, there is an improvement in the color, the patient who is unconscious becomes restless, there is an improvement in muscle tone, in venous tone and in the return circulation.

You can administer this with no fear of toxicity, because if toxicity occurs, you are getting the physiologic effect that you want to obtain. You want to get a muscle tone and push the blood back into circulation. You want to get your venous circulation going. If toxicity occurs, the only thing you are going to get is a slight convulsion which will soon pass away. So to those of you who have not been using this drug, I am not advocating a particular thing except for the benefits which you will derive by the use of this remedy.

Dr. Farmer's presentation has been a wonderful thing. There is one thing that I would like to stress: That tannic acid should not be used on the face and on the hands, that normal saline in your compression dressing would give you the best results. Severe burns should have early skin grafting. Do not wait until a large amount of granulating tissue forms. I would also emphasize early mobility. Have the patient use his extremities early and get the muscle tone back to normal as soon as possible.

ALFRED W. FARMER (closing): I have been asked to detail the factors which I think have played a part in the improvement in the last three decades. Of course, the greatest improvement came immediately in 1925 on the use of tannic acid, and that we believe was due to tannic acid and due to the heightened interest in burns at that time.

Second, the last great drop which came in the mortality rates was since 1938, and I believe that is due to the early treatment we give the patient. As Dr. Palmer has said, we would treat the patient in the first twelve hours or twenty-four hours or forty-hours very intensively, for what may be called secondary shock or toxemia.

Dr. Wagner emphasized that patients with minor burns are now coming into the hospital. That was the reason we made some estimate of the surface area of burn, because that has always been the criticism of any figures which have come out in the literature concerning increase in mortality rates, or decrease in mortality rates, and it is absolutely necessary that one define what he is dealing with in the way of burns as far as surface area, if possible, and, if possible, depth, too; but that we find impossible in a paper of this kind.

Concerning the stay at the hospital, our patients remain in the hospital about two to three weeks, if the burn is not all the way through the skin; and if it is and they need skin grafting, we are able to skin graft them in four to six weeks after they have had tannic acid applied and removed. We have grafted people within three weeks of the time a tannic acid coating has been applied.

Referring to coramine which Dr. Palmer discussed, I do not know anything about it. We administer coramine to those patients who look very sick, but why, I do not know.

TRAUMATIC ARTERIAL ANEURYSMS OF THE PERIPHERAL ARTERIES*

PATHOLOGY, CLINICAL MANIFESTATIONS, DIAGNOSIS AND TREATMENT

MIMS GAGE, M.D.†

Diplomate of the American Board of Surgery

NEW ORLEANS, LOUISIANA

D
UE to our participation in the present worldwide conflict, opportunities for observation and the study of all characters and types of wounds will be unlimited. The opportunity to increase our knowledge of vascular pathology will be greatly enhanced by the inevitable traumatic injuries to the peripheral vascular tree. That vascular injuries both systemic and peripheral will occur in a rather high percentage of the wounded is revealed by the statistics of World War I. According to Matas,[1] 20 to 25 per cent of the wounded reaching the first post of surgical relief will present wounds of the vascular system. Therefore, those that survive the primary vascular injury without loss of limb, will have as a sequela to the vascular injury, traumatic arterial and arteriovenous aneurysms. In World War I, Makins[2] compiled 523 traumatic aneurysms from various British hospitals, chiefly from London, while Solomon,[3] in Germany, estimated that fully 1,400 traumatic aneurysms had been operated upon up until 1918.

The incidence of traumatic aneurysms, according to Matas's statistics, ranged from 1 to 2 per cent of the wounded. Therefore, the incidence of traumatic arterial aneurysms is very low, when one considers that the 1 to 2 per cent incidence includes both the arterial and arteriovenous types. If one considers only the pure traumatic pseudo-arterial aneurysm, the incidence will still be lower. There are many factors that are contributory to the low incidence of traumatic aneurysms. Where large vessels are injured, the individual receiving the injury usually succumbs before he can receive first aid treatment. In those cases in which early treatment can be successfully

given, the bleeding vessels in the majority of cases are immediately ligated. In a rather high percentage of these cases, subsequent infection or ischemic gangrene necessitates amputation. Therefore, in the patients who have early ligation of the injured vessels and in the more unfortunate who lose these injured extremities, aneurysms cannot occur.

Again the anatomical location of the vessel injured plays an important rôle in the development of an aneurysm as a sequela to vascular injury. In the arteries that are more or less fixed in their anatomical position by the surrounding tissue as well as by the numerous vascular branches, they cannot bend or be caused to move out of the path of the projectile, by the pressure produced within the tissue, following the explosive violence of the bullet or shell fragment as it is motivated through the subcutaneous tissue. Whereas when the vessel or vessels are embedded within very loose cellular tissue and the vascular branches are scarce or absent, the vessel can, so to speak, dodge the missile. Therefore, the latter vessels will escape the type of injury necessary for the production of an aneurysm.

The incidence of traumatic aneurysms also will vary according to the vessels involved. Thus Makins states that the highest incidence was in the femoral tract. His statistics relative to the frequency of traumatic aneurysms to the vascular tree were as follows: femoral, 35.2 per cent; popliteal, 17.6 per cent; carotid, 17.5 per cent; axillary, 12.2 per cent; brachial, 9.5 per cent; subclavian 6 per cent and the other vessels less than 1 per cent. The incidences as stated above correspond more or less to the fixation or flexibility of the vessels.

As traumatic injury to the peripheral vessels results in both the arterial and arteriovenous types of aneurysms, only the former will be discussed in this presentation.

Traumatic arterial aneurysms can be classified etiologically as follows: (1) those due to ·bullet or shell fragments of high explosive shells and hand grenades, (2) splinters of glass and bomb fragments (air raids), (3) those due to stab wounds such as knife, bayonet and sharp, knife-like splinters of glass, and (4) those due to fractures of the long bones that are in close anatomical contact with the main peripheral arteries. The wounds of the soft parts associated with the above types of injury are divisible into punctured wounds and opened and lacerated wounds, the punctured wounds predominating in those cases that develop aneurysms.

The wounds of peripheral arteries secondary to the above types of injury can be classified into four groups (Fig. 1): (1) those in which

a large segment of the artery is destroyed, (2) those in which the artery is completely severed with destruction of the two arterial stumps, (3) those in which there is only partial or incomplete sever-
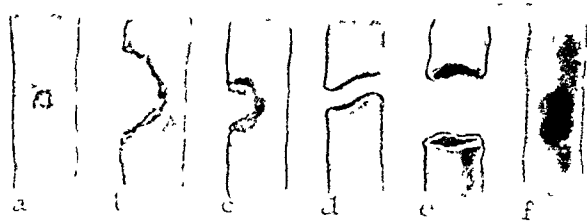
FIG. 1. Drawing showing the more common types of traumatic wounds of the arteries: (a) through-and-through wound; (b and c) lateral wounds with incomplete severance of the vessel wall. This type is dangerous due to the contracting longitudinal fibers which keep the wound open. (d) Complete severance knife wound; (e) destruction of vessel with complete severance, and (f) contusion of the arterial wall.

ance of the artery (lateral wounds), and (4) those in which the arterial wall is contused without primary loss of arterial wall substance.

In group one, in which a large segment of the artery is destroyed, the accompanying wound is usually an extensive lacerated and contused wound. The tissues about the artery as a rule reveal extensive destruction. This extensive destruction of muscles so frequently destroys the collaterals, that the majority of these patients bleed to death before aid can be given. If death does not occur immediately, and the individual reaches a first aid station, the vessels are ligated and the wounds débrided. Traumatic aneurysms seldom if ever develop in this type of injury. However, the percentage of ischemic gangrene necessitating amputations is very high. In group two a similar wound may be inflicted and the sequence of events take place as in group one. However, in group two if the wound of the soft parts is not extensive, bleeding may stop spontaneously by hematoma formation, which may give rise to latent aneurysm formation. Cases of this character, however, should be operated upon immediately, the devitalized tissue removed and the injured vessels resected and ligated. This prophylactic procedure will prevent the subsequent development of pseudo-arterial aneurysms. In group three there may be an open or even an extensive lacerated wound. If the latter is present, a formidable hemorrhage usually occurs and the life of the

individual, unless early treatment is available, is fatal in a large percentage of these cases. The procedure, of course, is similar to the treatment of one and two, early débridement and arterial ligation. However, the patients in this group who are prone to develop arterial aneurysms are those in whom the wound of the soft parts is a punctured wound. The wounds are usually produced by small fragments. They may be from bullets, splinters of shell fragment, high explosive bombs, hand grenades, and splinters of glass in civilian air raids. The hemorrhage is never severe in this type as hematoma formation is rapid and the pressure exerted by the hematoma in the surrounding limiting tissues results in pressure hemostasis. In the majority of these cases conservative treatment is the method of choice.

Conservatism is continued in the management of these cases, with constant investigation of and the evaluation of the efficiency of the blood supply to the tissues distal to the injury. There is one exception to this rule of conservatism and that is in the case in which the hematoma spreads into the tissues without limitation. When this undesirable complication occurs the bleeding into the extremity is excessive. The pressure of this extensive effusion of blood becomes so great that both the main artery and its collaterals as well as the venous system are completely blocked. The obvious results of this complication is gangrene of an extremity. This type of lesion demands immediate operation (provided shock is relieved as should be in all vascular injuries before any operative procedure is instigated) to stop the flow of blood by arterial ligation or suture and to relieve the increasing interstitial pressure which is of paramount importance. In group four, in which the artery is only contused, the incidence of aneurysms will be almost negligible. In the majority of cases of severe contusions of the arteries the intima is ruptured and intraluminal thrombosis invariably occurs very rapidly. The artery then becomes blocked at the site of contusion. Organization and re-cannulization rapidly follow in sequence, resulting in local strengthening of the vessel. However, where the contusion has been of less violent character, hemorrhage occurs in the wall of the artery. The bleeding seeps in between the muscle bundles and interstitial spaces, resulting in deficient localized nutrition and necrosis, the latter varying in its degree. It is, therefore, conceivable that this destructive process will precipitate a weak area in the arterial wall. Some weeks or months later this deficient area will stretch and balloon out producing a sacciform type of arterial aneurysm, or the wall may

rupture and produce a false aneurysm. This type of aneurysm has a high incidence of secondary rupture, due to the thinness of the damaged arterial wall. When this occurs, a secondary type of pseudo-aneurysm develops which has all the gross and clinical characteristics of the true traumatic type. Therefore, in all cases of wounds of the soft part in which the artery has not been severed but only contused, one should estimate carefully the direction and proximity of the wound to the peripheral vessels. If in one's opinion there has been arterial contusion (usually diagnosed by segmental spasm or throm-bosis resulting in diminished or absence of pulse below the site of injury), the case should be under constant observation for secondary arterial rupture and aneurysm formation.

In all cases in which there has been direct severance and destruc-tion of the arterial wall the concomitant vein is invariably injured. This converts both the arterial and venous channels into a common pool. When this occurs, an arteriovenous aneurysm is formed. The type of wound that is productive of arteriovenous aneurysms is in the majority of the cases a punctured wound. If injury to both artery and vein occurs in an open and lacerated wound arteriovenous aneurysms seldom if ever develop. It is the small punctured wound produced either by a bullet, small fragment of bomb, or shell, or splinters of glass that is responsible for the highest incidence of both arterial and arteriovenous aneurysms of the peripheral vessels. The high incidence of arteriovenous aneurysms following traumatic punctured wounds of the peripheral vessels is not within the scope of this discussion. However, attention must be called to their frequent occurrence in order that those called upon to treat vascular injuries in the present conflict will be constantly reminded of their occur-rence. They are more dangerous to life than are the arterial type, because of their severe local and systemic pathologic physiology, their operative difficulties and the hazards encountered in their surgical treatment and cure.

The pathology of traumatic arterial aneurysms varies consider-ably as regards the formation of their limiting walls and their peculiarities. Following the injury of an artery by a missile there occurs an immediate hemorrhage within the tissues which varies from profuse, moderate, to slight with or without external bleeding. The hemorrhage within the tissues may be divided into two types: (1) diffuse and progressive and (2) limited. Type one, as shown in Figures 3 and 5, occurs in areas where muscle planes form large spaces (popliteal) without limiting tissues. This type due to its

increasing pressure, compresses the artery at the site of the injury as well as the main artery distal and proximal to the injury including the collaterals. The incidence of ischemic gangrene in type one is very

Lateral wound of femoral artery

Extent of hematoma

Extent of hematoma

Complete severance of artery with destruction of wall
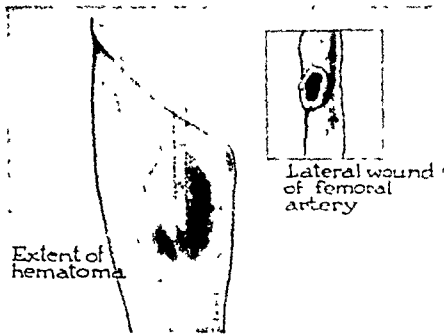
Fig. 2. Lateral wound of femoral artery with localized hematoma formation.
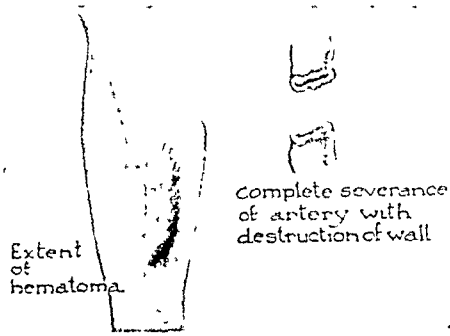
Fig. 3. Complete severance of femoral artery with large diffuse hematoma.

high, resulting not only in loss of the extremity involved but life as well.

Type two is that in which the extravasated blood is confined to a limited perivascular space by the surrounding musculo-aponeurotic planes resulting in a circumscribed hematoma. (Figs. 2 and 4.) This circumscribed hematoma may or may not pulsate synchronously with the heart beat. If pulsation is present, the lesion is then called a pulsating hematoma and is easily diagnosed. If, on the other hand, there is no pulsation, it simply signifies that the pressure of the hematoma is so great that it prevents escape of blood from the arterial injury. It is in the latter type that the aneurysm is delayed in its development for days or weeks following the initial injury.

Hematomas that are formed by vascular injuries may terminate in several ways. When the arterial lesion is small and the surrounding tissues limit the size of the hematoma by compression, the opening may be plugged by a clot. This stops the blood flow; organization follows with healing of the artery and absorption of the hematoma. The above sequence of events, however, seldom occurs either in military or civil practice, therefore, spontaneous recovery is the exception to the rule unless the arterial opening is of minute size. The hematoma may become infected, especially in those cases of punctured wounds, producing a phlegmonous process which varies in degree and extent depending upon the type and virulence of the invading organism. When a perivascular hematoma becomes infected, secondary hemorrhage invariably follows. It is most impor-

tant that the surgeon should be cognizant of the grave eventualities that may occur on opening these infected suppurating hematomas. Following the relief of tension there may occur a violent and uncon-

Stab wounds—
complete and partial
severance

Extent of hematoma
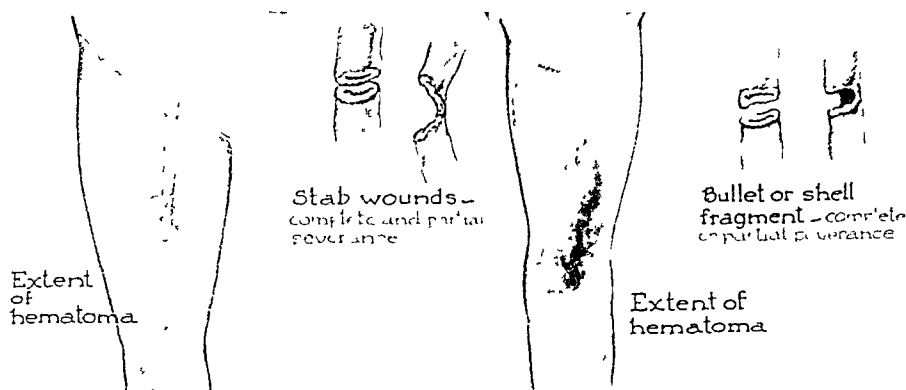
FIG. 4. Incomplete severance of arterial wall of femoral artery demonstrating a small firm localized hematoma.

Bullet or shell fragment—complete or partial severance

Extent of hematoma

FIG. 5. Wound of popliteal artery with extensive desiccating hematoma formation. This type of diffuse hematoma frequently compresses both the main artery and the collaterals.

trollable hemorrhage with sudden death. An infected hematoma associated with vascular injury should never be opened unless adequate precautions for the proximal control of the blood supply have been provided for in a most secure and efficient manner. This proximal control of the blood flow is of inestimable value in injuries of the large vessels at the root of the extremities and the base of the neck.

When the hematoma gradually spreads in the fascial and muscle spaces (popliteal and along the sciatic nerve to the gluteal region), the pressure produced by the blood clot increases rapidly in the tissues and compresses and obliterates the collaterals as well as the main vessel. When this takes place there occurs a rapid ischemia of the tissues and gangrene with loss of an extremity ensues. In other instances, the patient actually bleeds to death within his own tissues. There probably occurs a train of events that are produced by a loss of fluids plus total blood elements (shock resulting from leg trauma) that result in shock, which is progressive and irreversible, resulting in death. The author has seen two patients with fracture of the femur with tearing of the femoral artery who bled to death; the bleeding was entirely within the tissues of the thigh. There were no signs of external bleeding in either case as the fractures were not of

the compound type. In these cases of spreading hematoma it is absolutely imperative that immediate surgical exploration be undertaken. Delay in these cases always proves costly in that, not only is loss of limb to be expected, but life itself.

The small compressed and hard hematoma limited in its extent by aponeurotic structures, may cause sufficient pressure on the main arterial stem that it acts almost in the same way as an embolus. There occurs an abrupt localized obliteration of the arterial lumen, which inhibits blood flow distal to the obstruction. The collaterals do not have sufficient time for their development, consequently ischemia of varying degrees occurs. The resulting pathological condition ranges from focal muscle necrosis to segmental or massive gangrene. When these small hematomas compress and obliterate the main artery (usually the veins suffer the same pressure and obliteration), immediate surgery is indicated to release the pressure. The hematoma should be evacuated from the tissue and the injured vessel dealt with either by suture or ligation. In war surgery, ligation is probably the procedure of choice, because arteriorrhaphy is too great a time consumer. The results of arteriorrhaphy in the majority of instances are excellent, i.e., restoration of pulse distal to the suture. However, if arteriography is resorted to, one will be impressed by the greater majority of circular sutured vessels that show obstruction at the suture line, the return of the pulse distal to the arteriorrhaphy being due entirely to blood flow through the collateral circulation and not through a restored lumen at the site of injury. Therefore, I would not only suggest but approve and recommend the procedure of ligation instead of suture, in the great majority of cases of injury to the peripheral arteries. *In either ligation or suture, the sympathetics to both the upper and lower extremities, dependent upon the anatomical location of the wound, should be "blocked out" by repeated novocain injection until the circulation has been restored and maintained by the development of the collateral vessels.*[4]

From the foregoing discussion of injuries to the arteries with hematoma formation, one can readily understand that a comparatively large number of these patients will subsequently develop traumatic aneurysms. When an artery is directly injured by a projected missile, three things may occur which have a direct bearing on the type of sac that is formed. When the artery is only partially severed, the hematoma surrounds the site of the injury resulting in a traumatic aneurysm which, in the majority of cases, is of the sacciform type. It is in this type of aneurysm that the communication

from the artery to the sac is a small opening in the arterial wall. Therefore, for practical purposes, almost the entire wall of the artery is present and the aneurysm sac sits (so to speak) upon the wall of the artery. This is the ideal type for restorative endo-aneurysmorrhaphy of Matas. Incidentally, in this type the main blood supply to the distal parts is through the main artery. Therefore, the collaterals are infrequently developed in the cases of sacciform aneurysm.

When the artery is completely penetrated by a missile, which traverses one wall and exits through the other (a rather rare injury) the artery has two openings with an intervening arterial wall. This type of injury also gives rise to a sacciform aneurysm, the sac being bilocular. This type, although sacciform, is more prone to have some development of the collaterals but is not remediable by a restorative type of endo-aneurysmorrhaphy. The obliterative type of endo-aneurysmorrhaphy is, therefore, the procedure of choice. When the artery is completely severed by a missile or knife, a large intervening and surrounding hematoma is quickly formed. The majority of these cases lead to the formation of the fusiform type of traumatic arterial aneurysm. There is a great variety of sac formation in the above type, the character and extent of the sac corresponding to the limiting muscles and fascial planes. In some instances the sac may be oblong or spindle-shape on one side with a pseudopod projecting on the opposite side. In other words, the anterior wall of the aneurysm sac may be spindle or fusiform in shape, whereas on the posterior aspect the posterior wall may be sacciform. This is frequently true in the lower third of the femoral arteries near abductor foramen and also in the popliteal artery. In the popliteal artery the anterior wall of the sac is limited by the posterior aspect of the osseous structures forming the knee, whereas the posterior wall of the sac is limited only by the skin and superficial fascia. Therefore, in aneurysms of the popliteal artery the majority have the gross and clinical appearance of the sacciform type. Not infrequently in aneurysms of the popliteal artery the superior pole rotates posteriorly and downward and the inferior pole rotates anteriorly and upward. It is in the above type that the surgeon if not familiar with the pole rotation will invariably injure the proximal artery in opening the sac as it courses downward on the posterior aspect of the aneurysm.

The formation and development of the sac of traumatic aneurysms is most interesting both to the surgeon and to the pathologist. The artery having been partially, subtotally, or completely disrupted, is immediately surrounded by profuse bleeding and hema-

toma formation which disrupts the surrounding periarterial tissues. The escaped blood rapidly coagulates forming a mass which by its compression on the surrounding tissues and vessels acts as a hemostatic plug. The surrounding tissues are infiltrated with escaped blood and the spread of the blood coagulum is limited by the surrounding musculofascial planes. However, the blood from the injured artery continues to be pumped into and against this coagulated blood clot. The elastic blood clot is then molded and stretched by the systolic force of the escaping blood until the blood clot conforms to the shape of the surrounding muscles and fascia which act as a barrier against further spread. The blood escaping from the injured artery maintains a liquid state in the clot immediately surrounding the artery. Eddies and whirlpools are set up in this liquid center which have a tendency to core out and mold the inner blood clot into a definite cavity. The tissues, surrounding this blood clot with its coagulated periphery and liquid mobile center, proliferate rapidly producing an inflammatory reactive limiting membrane at the periphery of the hematoma. The proliferating tissues rapidly invade the periphery of the blood clot organizing the outer wall of the hematoma, thus forming a definite fibrous wall extending completely around the periphery of the blood clot. This formation takes place rather slowly and is usually accomplished within three to six weeks. While the outer wall is being organized, new layers of clot are laid down at the periphery of the cavity of the hematoma which is continuous with the opening or openings in the artery. Therefore, from without a connective tissue wall is formed while from within layers of new clots are formed, producing a laminated aneurysmal wall. As soon as the cavity of the blood clot is formed and lamination begins to take place, the endothelium from the wound or wounds of the artery begins to proliferate very rapidly along the inner surface of the cavity. This rapid proliferation and extension of the blood vessel endothelium continues until the entire cavity is waxed with shining endothelium. There is now a fibrous connective tissue wall (a true living membrane) reinforced with laminated blood clot which surrounds the endothelialized cavity connecting directly with the artery. A true pseudoarterial aneurysm is thus formed. The entire mass now reveals an expansile pulsation, which varies greatly in character and degree and is synchronous with the cardiac impulse. The traumatic arterial aneurysm differs from the true type in that the walls of the latter contain the elements of the arterial wall; whereas in the former the walls are composed of laminated blood

clot, encased in a fibrous capsule derived from the periarterial adventitious tissue, which contains only the endothelial structures from the injured artery. Therefore, traumatic arterial aneurysms are called false or pseudoarterial aneurysms and the spontaneous ones are called true arterial aneurysms. Their clinical manifestations, behavior and treatment are similar.

The clinical manifestations of traumatic aneurysms of the peripheral arteries can be classified into two groups: (1) immediate and (2) late; both of which can be further classified into local and regional. The immediate symptoms are those of trauma of the soft parts plus hematoma formation in the line of a peripheral artery. The hematoma formation varies greatly in different cases as shown in Figures 2, 3, 4 and 5. The hematoma produces a rather tense swelling at the anatomical site of the injury, i.e., thigh, popliteal space, calf, neck, supraclavicular space, axilla, arms and forearms. There is no pulsation of the hematoma at first, due to the interstitial pressure which is transmitted to the hematoma. The peripheral pulses may be absent, distal to the hematoma. This phenomenon previously was interpreted as complete block of the main vessel, by the pressure of the hematoma, and secondly, by thrombotic occlusion of the arterial lumen at the site of the injury. To the above must be added another explanation, which has seldom been mentioned in the recent or past literature and that is segmental vasospasm secondary to the arterial injury. (Gage,[5] Ochsner and DeBakey,[6] Leriche.[7]) The trauma to the artery precipitates vasospasm which gradually spreads over the vascular tree of the extremity involved. (Haimovici.[8]) If this condition is not recognized immediately and specific therapy instituted, grave and irremediable consequences ensue. The local signs as described above are the ones observed by inspection and palpation which in themselves are most suggestive of vascular injury. On auscultation one may or may not hear a soft blowing systolic murmur. The presence or absence of a murmur is dependent upon the fluidity of the hematoma and its exerted pressure upon the involved vessel. Sometimes the murmur does not manifest itself for hours or even days following the vascular injury. However, when once established, it is pathognomonic of arterial aneurysm. It is considered the one infallible sign of arterial aneurysm when present. Therefore, when one suspects an injury to a peripheral artery, this sign should be frequently sought for; and if the main peripheral vessel is partially or wholly severed, the observer will be rewarded by this distinct and characteristic audible auscultatory sound.

The bruit at first is soft and blowing in character but as the aneurysm sac gradually takes form, the limiting membrane contracting, the blood clot becoming desiccated and laminated, the bruit gradually changes until a loud whistling or harsh systolic sound is heard. This systolic murmur or bruit varies considerably depending upon the size of the vessel, the size of the aneurysm sac, its elasticity and above all the size and type of opening or openings in the associated artery. However, with the changes in sound and pitch of the bruit one feature is always constant, and that is the murmur or bruit regardless of type is constantly and specifically systolic in time. This is always pathognomonic of the pure arterial aneurysm. This type of arterial aneurysmal murmur should never be confused with the continuous hum or roar with systolic exacerbations which is so characteristic of arteriovenous aneurysms. The variations in murmurs of all forms of aneurysms is one of the most interesting chapters in vascular surgery.

The propagation of the murmur in arterial aneurysms very seldom extends any considerable distance. The murmur remains localized, being transmitted distally only for a very short distance. This is also in contradistinction to the murmur of arteriovenous aneurysms which is transmitted over a wide area both peripherally and centrally. There is, however, a cardiac murmur in arterial injuries that was described by Makins[2] during the last war. This systolic cardiac murmur was heard in thirty-seven out of a total of 180 cases of arterial injuries. Of these thirty-seven cases in which the cardiac murmur was present, eighteen were arteriovenous injuries and nineteen were pure arterial. Therefore, in all suspected vascular injuries or in all cases of wounds, the presence of a systolic cardiac murmur is indicative of a peripheral vascular injury. The presence of a vascular injury should, therefore, be diligently sought for, not once, but many times, until one is completely satisfied that an arterial injury is nonexistent.

The late signs are practically the same as those described above with the exception of those demonstrated by palpation. As the tissues proliferate and contract the hematoma becomes progressively smaller until a minimum is reached. This is due to the buffeting of the fibrous wall by intrasaccular lamination and desiccation of the blood clot which prevents further shrinkage. When this stage is reached, usually within six weeks to three months after the injury, a well developed and defined traumatic arterial aneurysm is formed. One now finds a definite expansile tumor in the line of the artery

involved. The expansile pulsation is systolic in time and is synchronous with heart beat. The tumor mass is compressible to a certain degree, depending upon the amount of the intrasaccular laminated clot. Where the latter is thin, the compressibility is great. When the intrasaccular clot is very thick the compressibility is almost negligible. Therefore, the compressibility and expansile pulsation depend entirely upon the thickness of the intrasaccular laminated clot. This certainly is not properly appreciated, with the result that many cases of arterial aneurysm are not diagnosed.

Another sign that is of great diagnostic importance is the one in which the aneurysm decreases in size and stops pulsating when the proximal artery is obliterated. When the proximal artery that enters the aneurysm is compressed, the sac gradually decreases in size and all pulsations cease. If one auscultates the aneurysm while the proximal artery is compressed, no sounds are heard. The above signs are the most convincing proof of the presence of an arterial aneurysm.

Still another sign of less importance than the above is the diminution of the size of the aneurysm sac on elevation of the extremity in which it is located. As this only applies to aneurysms in the extremities, its usefulness as a diagnostic procedure is limited.

The regional symptoms are few and pertain mainly to the vessels distal to the point of arterial injury. If the arteries pulsate normally distal to the injury, it signifies that the blood supply to the tissues is through the main arterial channels. When the pulsation of the arteries is diminished, it is demonstrative that some of the blood flow is being shunted through the collaterals. If there is no palpable pulsation in the arteries distal to the injury, one is justified in assuming that the blood supply to the t'ssue is mainly through the collateral vessels. The latter condition is known as Delbet's sign which is of great prognostic importance because it indicates that an adequate collateral circulation has been developed. The successful cure of the majority of arterial aneurysms depends upon an adequate and sustained collateral circulation. Because the main artery must be obliterated, the importance of the above sign is readily understood. Occasionally, other peripheral signs manifest themselves in the form of some of the more common diseases of the peripheral arteries. When an embolus breaks off from the clot in the aneurysm sac, it frequently blocks one of the peripheral arteries. When this occurs, signs and symptoms are produced which simulate Buerger's disease. In some of the cases the sudden blockage of the artery distal to the aneurysm by an ambolus from the sac results

in gangrene of the tissues. The extent of the gangrene varies considerably in degree from small areas of necrosis to segmental and massive gangrenous processes. Therefore, one is occasionally confronted with a case of arterial aneurysm with signs and symptoms of Buerger's or Raynaud's disease. The aneurysm in the above type of case frequently goes undiagnosed. All cases presenting signs of peripheral arterial disease of the lower extremities should be investigated for aneurysms of the peripheral arteries. Recently the author saw a case which had been diagnosed as bilateral Buerger's disease of the lower extremities. There was gangrene of the fourth and fifth toes of the right foot with reddish cyanosis of both feet. On careful examination it was discovered that the patient had bilateral popliteal arterial aneurysms. The gangrene was due to embolic occlusion of the anterior tibial artery. The embolus had its origin from the thrombus within the aneurysm sac. The above case is cited to emphasize the ease with which serious mistakes in the diagnosis of aneurysm can be made.

Before any operative procedure can be applied to the traumatic arterial aneurysms one must first determine the presence or absence of an adequate collateral circulation. This is necessary because a successful operation for the cure of an aneurysm depends primarily on the presence of a collateral blood volume flow, sufficient to maintain normal metabolism to the tissues distal to the aneurysm, because the main artery will be obliterated at that site. If the collateral circulation is not adequately developed, gangrene will occur in from 5 to 15 per cent of the cases. Therefore, the first thing to do is to test the collateral circulation in all aneurysms of the peripheral arteries. This is best accomplished by the Matas method. This method has been repeatedly described by Matas[11] and the author.[4,5] The test is performed as follows: In the extremities a rubber bandage is applied from the toes to just above the aneurysm. This milks all the blood out of that part of the extremity covered by the bandage. The Matas compressor is now applied to the main artery just above the point where the rubber bandage stops. The artery is obliterated by the compressor and the rubber bandage quickly removed. The time factor between the removal of the bandage and the appearance of a florid blush to the cadaveric tissue demonstrates whether the collateral circulation is abundant or deficient. If adequate, the blush of the tissues occurs within three minutes. If deficient, the time element is longer; therefore, the greater the time element the less efficient is the collateral circulation. If the test demonstrates that

the collaterals are not developed, the operation must be postponed until the collaterals can be developed.

There are two methods that can be used to develop the collateral circulation: (1) the mechanical and (2) the physiological. The mechanical consists of daily application of the Matas[11] compressor, increasing the time of compression each day until the collaterals are developed. The physiologic[4,5] consists of removing the sympathetic vasomotor control from the peripheral arterial tree. This is accomplished either by mechanical or chemical section of the regional sympathetic ganglia and their connections. This method is preferable to the others because it can be accomplished in three to five days, whereas the other methods require weeks and sometimes months. The rationale of sympathetic block in the developing of the collateral circulation is based upon experimental data collected from the literature as well as the author's personal experience in peripheral vascular disease, including aneurysms. The fundamental principles involved are as follows: (1) overcome vasomotor spasm, (2) produce vasodilatation, (3) increase blood volume flow through the capillaries, (4) increase blood volume flow through the main arterial channel distal to the ligation, (5) increase the size of the vasa vasorum, (6) increase lymph flow, and (7) prevent ischemic gangrene. All of the above conditions occur within three to five days following sympathetic block with novocain and alcohol. The collateral circulation will be adequate and be sustained postoperatively which will prevent the occurrence of ischemic gangrene. Therefore, when the collaterals are developed and efficient, the aneurysm can be safely operated upon.

In the carotid group, the compressor is applied to the carotid to test the efficiency of the collateral circulation to the corresponding cerebral hemisphere. The carotids cannot be safely ligated until the patient can stand obliteration of the carotid for at least one-half hour. To develop the collateral circulation for the carotid, a combination of both the mechanical and physiologic methods is used. The stellate ganglion can be injected from the anterior or posterior approach. The physiologic method is also advocated for aneurysms of the subclavian, axillary and brachial arteries.

The surgical treatment of all arterial aneurysms of the peripheral arteries is based upon the sound principles promulgated by Matas[9] in 1903. He described at that time a new type of operation which revolutionized the surgical treatment of aneurysms of the peripheral arteries. The operation consisted of intrasaccular closure of the

arterial mouths feeding the sac, with obliteration of the aneurysm
sac. The operation was described as an endo-aneurysmorrhaphy.
He divided the operations into three types: (1) the restorative, (2)
reconstructive, and (3) the obliterative endo-aneurysmorrhaphy.

The restorative type is applicable only to the true sacciform
aneurysm where over two-thirds of the vessel wall is intact, the
opening in the artery being produced by a small lateral wound of the
vessel. In this type the sac is opened and the small opening closed
by interrupted sutures of silk or cotton. The excess sac is then
removed and the remaining portion used to reinforce the arterial
wall at the site of the sutured artery.

The reconstructive type of endo-aneurysmorrhaphy was advo-
cated for fusiform aneurysms. The technic was to construct a new
channel out of the walls of the sac. It was soon discovered that this
procedure was limited in its scope and was finally abandoned because
of the high incidence of postoperative thrombosis in the newly
created channel.

The obliterative type has become universally applicable to all
types of arterial aneurysms of the peripheral arteries except the pure
sacciform type. The procedure is of the greatest simplicity. It con-
sists simply of opening the aneurysm sac and closure of the arterial
openings of which there may be several, then obliterating the sac.
All suture material should be either cotton or silk, as absorbable
sutures will give way with dire consequences. In performing the
operation it is not necessary to dissect out the sac or either the
proximal or distal arteries; one simply exposes the anterior or
posterior wall of the aneurysm. The sac is then incised and the edges
retracted widely, thoroughly exposing the interior of the sac. All
clots are now removed, the sac cleansed thoroughly, and then
inspected for the arterial openings. It must be insisted upon that the
entire inner surface of the sac be inspected most carefully because
if one fails to do this, one or more small openings may be overlooked.
Each opening found is closed with interrupted silk or cotton sutures.
When all openings have been closed, the tourniquet is released to
see if any of the sutured openings leak. This is most important
because if the operation is finished before the removal of the tourni-
quet, bleeding may occur, even another aneurysm may be formed.
If no bleeding from the sac is encountered following removal of the
tourniquet, the sac is obliterated and the wound of the soft parts
is closed by interrupted sutures of cotton. This operative procedure,
as described by Matas, is simple in its execution, does not interfere

with the perisaccular collaterals, obliterates the vessels entering into the formation of the aneurysm and destroys the sac. It is, therefore, the ideal operation for aneurysms of the peripheral arteries.

The operation of Matas is, however, not applicable to all aneurysms of the peripheral arteries if we include the carotids, subclavians, the external iliacs and the common femoral. Aneurysms of the above arteries in the majority of instances are best treated by proximal polar ligation, Anel's operation. Polar ligation of the proximal artery is used mainly for aneurysms of the carotid and subclavian groups. Ligation of the subclavian and innominate artery proximal to the aneurysm offers considerable technical difficulties when approached from above. To overcome these difficulties, Touroff[10] recommends that the subclavian be ligated transpleurally. He reported a case of congenital arteriovenous aneurysm of the left upper extremity in which he ligated the first portion of the left subclavian artery transpleurally. He recommends that in traumatic injuries to the subclavian they be treated by transthoracic, transpleural ligation. The author subscribes to this operation and has successfully ligated the left subclavian, transthoracic-transpleural, for an arteriovenous aneurysm of the first portion of the left subclavian. This same procedure is recommended by the author for aneurysms of the right and left subclavians and the innominate and the low common carotids. However, for aneurysms of the first cervical portion of the common carotids, distal polar ligation has been the operation of choice. It would be preferable to use the approach by Touroff.[10]

In traumatic aneurysms involving the external iliac and common femoral the transabdominal ligation of the external iliac is the procedure of choice.

REFERENCES

1. MATAS, R. Military surgery of the vascular system. Keen's Surgery. Vol. 7. Philadelphia, 1921. W. B. Saunders.
2. MAKINS, G. H. Gunshot Injuries to the Blood. Baltimore, 1919. William Wood.
3. SOLOMON. Quoted by Matas.[1]
4. GAGE, MIMS. Arterial aneurysms of the peripheral arteries. Tr. South. Surg. Ass., 52: 257, 1940. Idem. Mycotic aneurysms of common iliac artery. Tr. South. Surg. Ass., 46: 473, 1934.
5. GAGE, MIMS and OCHSNER, ALTON. The prevention of ischemic gangrene following surgical operations upon major peripheral arteries by chemical section of the cervicodorsal and lumbar sympathetics. Ann. Surg., 112: 938, 1940.
6. OCHSNER, ALTON and DEBAKEY, MICHAEL. Thrombophlebitis and phlebothrombosis. South. Surg., 8: 269, 1939. Idem. Therapy of phlebothrombosis and thrombophlebitis. Arch. Surg., 40: 208, 1940.

7. LERICHE, RENE. Les malades des ligatures moyens de les prévenir et de les traites. *Presse méd.*, 1: 41, 1940.
8. HAIMOVICI, H. From a review of the 46th French Congress of Surgery, October 4, 1937. Trans. DeBakey, Michael. *Surgery*, 3: 306, 1938.
9. MATAS, R. Surgery of the Vascular System: Aneurysms: Keen's Surgery. Vol. 5, p. 216. Philadelphia, 1920. W. B. Saunders. Idem. An operation for the radical cure of aneurysms based upon arteriorrhaphy. *Ann. Surg.*, 37: 161, 1903.
10. TOUROFF, A. S. W. Transthoracic, transpleural ligation of the first portion of the left subclavian artery. *Surgery*, 10: 747, 1941.
11. MATAS, R. Testing the efficiency of the collateral circulation as a preliminary to the occlusion of the great surgical arteries. *Ann. Surg.*, 53: 1, 1911.

DISCUSSION

LOUIS G. HERRMANN (Cincinnati, Ohio): Dr. Gage has told us many of the important problems concerning the injury to major arteries in the extremities. He has talked largely about gunshot wounds and stab wounds, and I think we all now believe that reconstruction, either by the Matas' procedure or by Dr. Gage's, is by far the best method of dealing with such things. However, I would like to add one thing, and that is with regard to the cure of aneurysms which result from disease of the wall, especially the syphilitic aneurysms that develop in the popliteal space. It is extremely difficult, and sometimes even dangerous, to attempt to reconstruct them or obliterate them by the Matas' procedure, so that we as a rule attempt to open the sac with the supply of blood controlled by a tourniquet, and then go outside of the sac and isolate the incoming and outgoing arteries, and ligating them high above and high below the sac rather than try to obliterate them at the sac itself.

We have had some recurrences when we have tried to obliterate them by the Matas' procedure alone.

There is very little else to add to what Dr. Gage has already told us. I think he has shown us very definitely that at least traumatic injuries to blood vessels should be repaired with nonabsorbing materials. I think most of the experimental work now will substantiate the fact that catgut, although it does not in many cases, is more or less a dangerous type of suture to use, and that either silk, cotton bandage, cotton tape or some such nonabsorbent material should be used.

There are many factors that Dr. Gage did not have time to touch upon. That is, there are other methods, of course, of developing collateral circulation, and the test that Dr. Gage has described, namely, Dr. Matas' test, is an extremely valuable one; and we believe very definitely that no patient should have a large peripheral artery ligated unless we are certain that the collateral circulation is capable of carrying on.

A little trick that we frequently use for a day, two or three days, or maybe a week, is to insist that the patient compress that artery with his finger in order to promote a more efficient collateral circulation. In young patients and in healthy patients, such a collateral circulation could be developed without mechanical means. The blocking of the sympathetic

chain is a simple thing, as Dr. Gage has told us, and is a very valuable thing. I would stop, however, with the novocaine and not inject the alcohol, but that is just a matter of personal opinion.

The other means of developing collateral circulation which we use in our own clinic have proved valuable and in the last ten years we have not lost any extremities after ligating large vessels, including large vessels of the popliteal space. It is fairly simple to deal with a large aneurysm of the sac in the popliteal and in the femoral region. You control hemorrhage first by tourniquet and then ligate above and below the sac and then ligate the collaterals under there, or use a group of sutures and obliterate the incoming and outgoing vessels in the sac itself.

EDGAR L. GILCREEST (San Francisco, Calif.): Dr. Gage has given us one of his clear, concise papers which I think we are all indebted to him for, particularly at this time. I believe in this great emergency, when we are going to have literally hundreds of doctors dealing with aneurysm, that the admonition of Halsted probably should be publicized, so that surgeons will bear in mind the common errors that he called our attention to years ago. I shall read seven of them.

"1. Opening the sac or pulsating hematoma without first making a temporary occlusion of all the possible sources of hemorrhage.

"2. The permanent ligation of a great arterial trunk as a precautionary measure in the search for a distal bleeding point.

"3. Ligation of a trunk too far from the aneurysm.

"4. Stuffing the wound with gauze to arrest hemorrhage.

"5. Drainage.

"6. The employment of catgut for the ligature, or of silk that is too fine.

"7. Ligation of the artery proximal to an arteriovenous aneurysm or fistula."

In 1929, in a paper on aneurysm, I closed that paper with this remark: "Surgeons attempting to intervene in aneurysms or arteriovenous fistula, should be much more concerned with hemostasis and meticulous technic than with the duration of the operation. These operations are at best always tedious and time-robbing and demand the best of the surgeon's skill. Without producing hemorrhage, these patients stand operations of six or seven hours' duration without severe shock."

And then I quoted Halsted again when he said, "An operator searching for a bleeding point in a pool of blood, and particularly so when embarrassed in his movements by the adherent walls of an aneurysm within which he is working so disadvantageously, presents a distressing spectacle." He said, "I would rather devote an additional hour or more to an operation than be caught for a few moments in such a predicament."

I should like to present a case of an arteriovenous fistula. This patient was walking down the street in Mexico City and was shot by a bandit, the bullet entering the right clavicle. That happened in December, 1920. The

arm did not swell much until early in 1927. During that time, no one had ever told him to elevate his arm, even at night. After he had elevated the arm for six weeks and gotten the edema out of the arm he was operated upon. At the operation I removed one of the clavicles, which is so important. It was Thompson of Galveston who perhaps contributed much on this subject, and he was one of those who has called our attention to the great importance of ligation of the first part of the subclavicle. I think that was due to the fact that in those days, they did not remove the clavicle, which makes this operation much easier.

In this operation I ligated first the innominate vein, the subclavian and jugulars. The five ligations done at that time were: the subclavian artery, the subclavian vein in two parts, the external and internal jugular vein and innominate vein.

During that operation, after we made those ligations, the thrill became imperceptible and the bruit became inaudible, and I went out among the adhesions and ligated what we thought was another portion of the subclavian, and we believed we had better stop at that time.

Nine months later he returned because his arm had begun to swell a few weeks before. We had expected that might occur and realized that we must go then way out and do further ligations. The various ligations made in the first operation and in the second operation numbered eleven in all. Dr. Emile Holman showed the necessity of ligating the innominate vein and preventing gangrene in the extremity.

COMMANDER RICHEY L. WAUGH (Boston, Mass.): I want to add just a few more words about the fistula. We have found in treating the traumatic wounds of the upper extremity quite a few of the arteriovenous fistulas. Even during the past three months, we have had two cases, and I would just like to take a minute to refer to those two cases. Both were due to missiles, one a chip fragment occurring to a young man who was working at the Watertown Arsenal, and the other followed a gunshot wound, rather late after the injury.

In the case of the young man who had a chip fragment enter the fore-arm, within one week he perceived a warmth and a thrill in the forearm. We operated upon this patient and found an arteriovenous fistula between the ulnar artery and the deep ulnar vein. The treatment was, of course, a quadruple ligation.

In the other case, the gunshot wound in the arm occurred ten years previously. It was followed about four years ago with a severe contusion in which there was ecchymosis and soreness for several days, and I believe only a year ago was the patient aware that there was something wrong with the extremity. He noticed, whenever he put his face on his arm, that it felt much warmer. We operated upon this patient.

First, may I say that in both of these cases, with digital compression over the site that would stop the fistula, there was a slowing of the pulse from ten to fifteen. That is known, I believe, as a Branham's sign.

We operated upon the patient and found a fistula between the profunda artery and the vein. This patient also was treated by quadruple ligation. He is now in our hospital convalescing.

JOSEPH E. J. KING (New York, N. Y.): Since Commander Waugh spoke of the arteriovenous lesion, I should like to make a brief remark on the handling of those lesions as proposed by Dr. Matas. We do know that in the last war we had several cases, and I refer especially to the femoral region above the profunda. Those legs had lesions and instead of doing what Dr. Matas later told us to do, we did quadruple ligation. Fortunately, all the legs were saved, but it is so much better to ligate the vein, open it up, sew the opening in the artery, preserve that section of the vein and sew it over to a patch instead of a quadruple ligation. We did not know that during the last war, and I should like to call this to your attention because you may have to do it.

MIMS GAGE (closing): I am sorry I did not have time enough to go into the pathology and the clinical manifestations in the syphilitic type of aneurysms in speaking of the traumatic aneurysms, but to do that would be too time-consuming.

As regards catgut, Dr. Matas advocated many years ago that we not use it because he had had many of those sutures break down.

As far as development of intravenous occlusion in the subclavicle is concerned, I said in civil practice it takes time. It takes from six weeks to two or three months to develop intravenous occlusion by the Matas compressor or Dr. Herrmann's Pavex machine. The sympathetic block happens to be my child and I have to nourish it along over a long road. It does save time because you can block the aneurysm one day and operate upon it the next.

I have had over ten popliteal aneurysms, all syphilitic but one. In that case I obliterated his arteries secondary to block. I have not had any gangrene in those cases.

Most of us have had syphilitic popliteal aneurysms and have had no trouble suturing them.

I do not know of anything, as Dr. Gilcrest has said, that the surgeon is confronted with that gives him more trouble than an arteriovenous aneurysm at the roots of a limb, including the neck if you want to include that as a limb. You have to control not only the artery and the vein on the proximal and distal side, but all the collaterals as well. Very frequently up in the neck arteriovenous aneurysm defeats you; it really whips you down; you cannot make it. I had one I worked upon for two hours and could not do a thing and had to do a transthoracic ligation of the artery.

The majority of arteriovenous aneurysms have to have quadruple ligation.

The thing I wanted to stress is that during the war we are going to have a large number of injuries to the peripheral arteries and I think if the sur-

geons will recognize them as such and operate upon them as soon as they possibly can, ligate the arteries and then turn them over and block the sympathetics with novocaine, they will save many of these limbs. You can do it without alcohol, but you have to repeat that every day for eight to ten injections until the collaterals are developed. You have no time to carry a Pavex machine or a Matas compressor around with you. If you determine that the artery is injured—which you usually can—it should be ligated and the development of an aneurysm prevented. When an aneurysm manifests itself within ten days to two weeks after the injury, it should never be touched until three to six months later in order to let all the tissue become organized. That is a law that Dr. Matas stated many years ago; because if you operate too soon when the tissue is friable, your sutures will not hold, with the result that a disaster will occur in a giving-away of the suture line.

As far as the operation is concerned, it is simple, provided you can control the proximal blood supply. Where you cannot, you have to dissect out the entire affair and control the blood supply below and above by using temporary ligatures. Outside of that, it is simple; but arteriovenous aneurysm is an entirely different chapter and would require too much time.

FROSTBITE IN SHIPWRECKED MARINERS*

G. M. BROWNRIGG, M.D.

Assistant Surgeon, St. John's General Hospital; Surgeon, Sanatorium, St. John's

ST. JOHN'S, NEWFOUNDLAND

D URING the past eighteen months I have been one of a group of St. John's surgeons privileged to share in the surgical care of a large group of shipwrecked mariners, survivors of the Battle of the Atlantic, who have been landed at our port.

Of this group 75 per cent of all those who required hospitalization suffered from lesions of the extremities produced as a result of exposure to cold and immersion in the icy waters of the North Atlantic. Many of them had been adrift in open boats or on rafts for periods of up to ten days and some had been completely immersed in the water before being taken into the boats.

No definite figures on the degree of cold to which these men were subjected can be, under the circumstances, expected to be available. We do, however, have reliable data on both atmospheric and water temperatures in the areas where these men were exposed during the winter and early months of spring. Water temperatures in these regions vary from 28° to 30°F. in the more northerly regions and up to 35°F. in the more southerly. The atmospheric temperature naturally shows wider variations but in the wind an atmospheric temperature of 0°F. is not uncommon. It is thus obvious that these men were exposed to atmospheric temperatures often below freezing point and to water of a temperature just about freezing point.

In civil practice frostbite has until recently excited but little comment. Brahdy,[18,19] however, reported no fewer than 996 cases as occurring during the years 1933 to 1936 amongst outdoor municipal employees of the City of New York. Successive military campaigns have directed considerable attention to this problem. During the Napoleonic Wars Larrey[1] observed numerous cases. The condition was adequately described during the Crimean War[2] and later by Page[8] during the Balkan Wars. There was some confusion at times as to the etiological factor[8] but in every case they were ultimately recorded as frostbite. During the last Great War large numbers of

cases occurred and the name "trench foot"[9] was applied to some of these cases.

Other terms have also been applied. Liggett[20] used the term "dermatitis congelationis." Recently, Webster, Woolhouse and Johnston[27] and Greene[29] have applied the term "immersion foot" to describe lesions present in a group of cases similar to our own. I quote from the excellent study of Smith, Ritchie and Dawson[10] upon the pathology of "trench foot": "The applicability of the term frostbite to the condition to be inquired into has been questioned. After consideration we have decided to continue the use of the word as it accentuates the essential causal factor concerned, namely, cold, and, at the same time, is sufficiently comprehensive to include the various local effects which cold may produce."

We turn now to our own cases. It was soon apparent that these men suffered from lesions of varying degrees of severity and that they could be divided broadly into three groups depending upon the type of lesion presenting. The lower extremities were in all cases affected. In a few cases the upper extremities were affected but never independently of the lower. The lesions were not symmetrical in all cases, that is to say, there were instances in which one leg presented what we shall describe as first degree frostbite, and the other what we shall describe as second degree frostbite; and there were still others with second degree frostbite on one side and with third degree frostbite on the other.

TABLE 1

Survivors Requiring Hospitalization	No. of Cases of Frostbite	Percentage with Frostbite
94	71	75

Nearly all patients, irrespective of the group to which they belonged, gave a similar history. They had noticed no pain until they had been rescued. They had been able to move about, so far as movement was possible, in the life boats, but when rescued or shortly after had lost the use of their legs and experienced pain of varying degrees of severity. Some of them had worn ordinary footwear, some only socks, but the presence or absence of footwear did not appear to have any influence upon the occurrence or severity of the lesions. The history of the onset is similar in all respects to that described by Page[8] in the cases seen by him in the Balkan Wars,

Group I. (*55 Cases.*)   Some of the cases in group one were able to walk upon admission. They complained of pain in the affected extremities. The feet and legs were swollen, usually to but not above the junction of the middle and lower third of the leg. There was some erythema but otherwise no generalized color changes were noted. All movements were present. The extremities were warm or hot to the touch. Some areas of anesthesia were noted in the feet but these disappeared after admission. We did not study the distribution and extent of these areas.* Circulation was apparently unaffected and palpable pulsation was present in the dorsalis pedis and posterior tibial vessels. There was in many a slight temperature upon admission of 99 to 100°c. which usually subsided within a few days.

TABLE II

No. of Cases of Frostbite	First Degree	Second Degree	Third Degree
71	55 77%	7 10%	9 13%

TABLE III

No. of Cases	Lower Extremities Affected	Upper Extremities Affected
71	71 %—100	8 %—11

In many of the cases small areas of dusky red mottling were present and bleb formation was noted in many of these areas. The blebs commonly contained dark bloody fluid, in some, straw colored fluid. These areas of mottling and bleb formation occurred most frequently on pressure points; thus the metatarsophalangeal joint of the great toe and of the small toe, the malleoli and heel, and the dorsum of the toes were the parts most frequently affected in this manner. This, I think, is of considerable importance when we come to consider treatment.

Group II. (*7 Cases.*)   The cases in this group showed a condition similar to the Group I cases but in addition more serious lesions were present. One or more toes was dusky red in color or black; movement in the toe or toes was absent; it was cold to the touch and sensation

---

* These areas of anesthesia might possibly be due to inflammatory changes in the end organs.

was absent. In shoor, areas of dry gangrene were present affecting chiefly the toes, but in some the more distal areas of the foot were affected.

Group III. (*9 Cases.*)   The cases which fall under this group presented a picture similar at first to the cases in Group I and Group II. The lesions described as proper to Group I and Group II were always present but in addition there were important differences. Usually larger blebs were present and these seemed less superficial and occurred higher up in the affected extremity. One important difference was obvious: From the time they came under observation they appeared toxic and a high fever was present from the onset. Temperatures of 103 to 104°c. were the rule and we soon came to realize that a high fever, in the absence of intercurrent respiratory infection, was significant of grave tissue damage. When I reviewed the cases this impression was confirmed. Every patient with a high fever on admission proved later to belong to Group III.

Not long after admission other signs made their appearance which obviously differentiated these cases from those in the other groups. While cases in Group I and Group II rarely showed new areas of mottling following the institution of treatment, the patients in Group III did; and in spite of rigid aseptic precautions, evidence that infection existed soon became obvious. The parts were hotter to touch and more reddened in appearance. Deep, foul-smelling areas of slough developed; tendons and muscles became, in the course of time, exposed and a foul, putrid, offensive odor developed. Thus, a type of wet gangrene was established in the soft tissues of the extremities. Even in these cases pulsation was present in the large vessels and secondary hemorrhage, though never of serious degree, often was present.

I am convinced that this infection was not introduced into the tissues but was present from the onset as evidenced by the fever often present before any break in the surface tissues occurred. This impression is dependent in part upon and is confirmed by the pathological findings of Smith, Ritchie and Dawson.[10] Further it was noted that muscles and tendons sloughed and became necrotic and exposed but there was notable absence of abscess formation in the tissue spaces.

The upper extremities did not in all cases escape involvement but in no case were the lesions observed in the upper extremities more serious than the type of lesion classified as Group I in the lower extremities.

I should like at this point to draw attention again to several facts. The least serious cases showed in the extremities swelling only with or without bleb formation. These lesions were also present in more serious cases. In the more serious cases in Group II, there was in addition mummification of the most distal areas of the extremity, that is to say, in the areas where the extremity had the least mass, and in the most serious cases, in addition to the Group II lesions, there were present lesions affecting the deeper and thicker tissue masses more proximally in the extremity. Thus it seems to me that we can visualize the sequence of events as follows: The first response was swelling in affected parts. Where there was continuation of exposure beyond this stage the most distal parts, where the mass surface ratio is least, where heat loss to mass ratio can therefore be greatest and where the effects of cold can penetrate to a sufficient depth to produce a maximum reaction, the tissue response was sufficiently great to interfere completely wih tissue nutrition and dry gangrene resulted. Further exposure resulted in damage to structures higher up, chiefly tendons and muscles, of a degree sufficient to bring about necrosis of tissue; but because the thickness or mass of the tissues provided a degree of protection to the cold to which they were subjected, this process was necessarily not so complete as in the case of the more distal parts. Therefore, the death or gangrene of the tissues was slower in onset and at first less complete and so produced what we are pleased to call wet gangrene.

It is my belief that this wet gangrene is not, as some suppose, primarily an infection in the tissues but necrosis due to a physical agent, namely, cold, in which infecting organisms played a saphrophytic rôle. I must again refer to the work of Smith, Ritchie and Dawson[10] to explain the presence of organisms in the tissues. I should like to stress here again the fact that where Group II lesions existed that were present upon admission and where Group III lesions occurred they, too, were present upon admission as evidenced first of all by a high fever. In other words tissue responses and tissue damage were dependent upon exposure, not upon subsequent events. This observation is not a new one and was first noted by Page.[8]

I believe that it would be of inestimable value both to the statistical study of these cases and to a proper understanding of the prognosis in the individual case if we were to classify them somewhat as we classify damage caused by heat, according to the degree of tissue response and damage.

Thus I would suggest that where exposure to cold is the etiological factor we retain the term frostbite. Where the lesions resulting correspond to Group I in our cases we should classify them as frostbite, first degree; where Group II lesions are present we should classify them as frostbite, second degree, and where Group III lesions are present, frostbite, third degree. We would thus be able to say that in our cases we saw frostbite of first, second and third degree in the lower extremities, and of first degree only in the upper extremities.

That this classification, at least as regards first and second degree frostbite is applicable to ordinary cases seen in civil practice, is confirmed by study of the types of cases described by, Brahdy.[18,19] In civil practice we have seen one case of frostbite where dry gangrene extended above the ankle joint and with wet gangrene above this area in the muscles. This lesion was produced by prolonged exposure to a more intense degree of cold. There might be some justification for classing this as fourth degree frostbite. As applied to prognosis there is little essential difference from our third degree cases. Loss of limb results.

A pathological study of tissues removed from patients suffering from frostbite is necessarily of limited value since it offers only a picture of the end results. There are, fortunately, several experimental studies of great value. Richspler[3] studied the effects of freezing on animal tissues. He found that the pathological changes which resulted from freezing were entirely inflammatory in nature and that there were not specific lesions of the vessels or nerves. Smith, Ritchie and Dawson[10] studied the effects of exposure to cold and wetness upon the shaven extremities of rabbits. They tried insofar as possible to reproduce the conditions under which "trench foot" occurred and their findings are obviously applicable to the cases in our series. Their findings were strikingly similar to those of Richspler. The degree of wetness or cold to which the tissues were subjected made no essential difference to the type of tissue response except quantitatively. Grossly, the tissues responded by edema. Sections taken shortly after its development showed enlargement of the endothelial cells in the intima of smaller vessels and infiltration of the perivascular tissues with inflammatory cells. Connective tissue cells were swollen and there was plentiful deposition of fibrin in the tissue spaces, molt marked beneath the skin papillae. Concomitant changes of an inflammatory nature were observed in the regional lymph-

nodes. Organisms, staphylococci and streptococci were observed in the subcutaneous tissues before any break in the integument occurred. Application of heat to or constriction of the damaged tissues appeared to be harmful and resulted in extravasation of blood. Sections* of tissues removed from patients in our series differed in no important respect from those described by Brahdy.[18]

Bearing in mind the pathology of frostbite, certain general observations on the early treatment of the condition may be made:

It is obvious from the experimental studies described above that trauma to the tissues must be avoided. Rubbing, with or without snow, is certainly harmful. Heat, in any form, must be avoided. This was first noted by Larrey[1] and Davys.[5] In recent articles Greene[24] and Clouston[23] have both stressed these points. There is a tendency by some, to use the Pavex treatment early in the disease. Greene[24] advises against this. On pathological grounds there are serious objections to its use since there is certainly a risk of additional trauma to the tissues. Miscall,[21] in 1937, suggested that paravertebral sympathetic block might be considered without, however, recommending it. Here again, I can see no possible advantage and upon pathological grounds there are, I think, serious objections. Certainly trauma and infection must be rigidly guarded against. In first degree frostbite, rest, elevation and strict attention to asepsis resulted in a rapid · amelioration of the condition in our hands. As soon as the cases came under observation this treatment was instituted and in no patient did we observe any progression of the disease while under treatment. Swelling subsided in due course; the pain gradually disappeared and in the majority there was complete or nearly complete restoration to integrity.

There were, however, some in whom a considerable disability persisted. This resulted from persisting thickening and stiffness of the toes, feet, and in one case, of the hands. It is obvious from the pathological data available that this is due to fibrous tissue formation. Since the formation of fibrous tissue is dependent upon tissue reaction it is equally obvious, I think, that anything which can minimize tissue reaction may be of considerable value.†[13,15] Webster,

* I am indebted to Dr. J. E. Josephson, Pathologist, Public Health Laboratories, St. John's, for his invaluable assistance in the study of these tissues. I am also indebted to Dr. W. Roberts for his kindly encouragement in preparation of the paper.

† Thus Lake suggested the use of vasoconstrictor drugs to reduce the head of pressure on the capillaries and Lewis and Love suggested obstructing the circulation in an effort to achieve the same results.

Woolhouse and Johnston[27] have recently employed and described the use of refrigeration of the affected tissues. They have noted immediate improvement following the institution of this treatment as evidenced by improvement following disappearance of the swelling and the reabsorption of blebs.

In the late treatment of these cases, that is to say, when the primary inflammatory reaction has subsided, physiotherapy, undoubtedly has a place and should be employed.

In the treatment of second degree frostbite conservatism should be practised. Rabut,[22] Greene,[24] and Thompson[26] all have observed that the line of demarcation may ultimately prove to be considerably distal to its apparent site. I can recall one case of frostbite, not in the present series, in which the distal phalanx of the finger appeared to be completely mummified. In the course of time the mummified area completely separated and fell off like a glove leaving a complete and normally functioning distal phalanx. In many cases it is obvious, if sufficient time is allowed to elapse, that superficially the area of gangrene is much more extensive than is present in the deeper tissues. In these cases in our series in which amputations were practised I did not observe that any significant time was saved, for in many healing was considerably delayed. It would seem that the ideal treatment would be to allow spontaneous separation of areas of dry gangrene, to trim necrotic bone and permit healing by granulation. This will in most cases serve to preserve the maximum amount of function.

In third degree frostbite amputation must be performed sufficiently high to permit removal of all areas of gangrene. We did not use guillotine amputations but in all cases flaps were cut and in no case was re-amputation necessary because of infection or spread of gangrenous process. There were, of course, some instances in which small areas of slough developed in the skin but these did not prove serious. Since these amputations were life-saving procedures which were performed upon tissues seriously devitalized, we did not think it proper to concern ourselves at the time of amputation with the ultimate problems of prosthesis.*

The problem of prevention of frostbite in shipwrecked mariners is admittedly difficult but it is my opinion that many of these cases can be prevented. I believe that the provision of a form of protection for the lower extremities at least, which would prevent heat loss

---

* Had dry gangrene been present, prosthesis would, of course, have been the factor in determining both the time and site of amputation.

and at the same time be waterproof, would be a great advancement. Soft sealskin boots might be of considerable value.

Sailors operating in submarine zones are now compelled to wear life-jackets at all times. This is fitted with a signal light. The addition of a waterproof compartment containing protective garments for the extremities should not prove impossible.

REFERENCES

1. LARREY. Memoires de Chirurgie Militaire. Vol. III, p. 60. Paris, 1812.
2. Medical and Surgical History of the British Army during War against Russia in Years 1854–1856. Vol. II, p. 187. London, 1858. H. M. Stationary Office.
3. RISCHPLER. *Beitr. z. path. Anat. u. z. allg. Path.*, 28: 451, 1900.
4. CONNOR, POWELL. *Indian M. Gaz.*, 39: 365, 1904.
5. DAVYS. Ibid, p. 245.
6. MACPHERSON, W. G. Quoted by Thompson. (1905). Medical and Sanitary Reports from British Officers attached to the Russian and Japanese Forces in the field. London, 1905.
7. WEITING. *Centralbl. f. Chir.*, 40: 593, 1913.
8. PAGE, C. M. *Brit. M. J.*, 2: 386, 1914.
9. SMITH, J. L., RITCHIE, J. and DAWSON, J. *Lancet*, 2: 595, 1915.
10. Idem. *J. Path. Bact.*, 20: 159, 1915.
11. SWAN, J. *Proc. Roy. Soc. Med.*, 8: p. 41, 1915.
12. FEARNSIDES and CULPIN. *Brit. M. J.*, 1: p. 84, 1915.
13. LAKE, N. C. *Lancet*, 2: 557, 1917.
14. MACKLIN, A. H. Treatment of frostbite. *Lancet*, 1: 884–885, 1925.
15. LEWIS and LOVE. *Heart*, 13: i, 1926.
16. HERMAN, L. G. and REID, M. R. The Pavaex (passive muscular exercise) treatment of obliterative arterial diseases of the extremities. *J. Med.*, December, 1933.
17. SHIPLEY, A. M. and YEAGER, G. H. Passive vascular exercise in the treatment of peripheral vascular disease. *Surg., Gynec. & Obst.*, 59: 480–485, 1934.
18. BRAHDY, L. Frostbite among employees of the City of New York. *J. A. M. A.*, 104: 529, 1935.
19. Idem. *J. A. M. A.*, 108: 369, 1937.
20. LIGGETT, H. Otoplasty for dermatitis congelationis (frostbite). *Med. Rec.*, 142: 278, 1935.
21. MISCALL, L. Frostbite. *Surg. Clin. North America*, p. 303, February, 1937.
22. RABUT, R. *Presse méd.*, 47: 1683, 1939.
23. CLOUSTON, H. R. *Canad. M. A. J.*, 40: 166, 1939.
24. GREENE, R. *Lancet*, p. 303, 1940.
25. DUCUING, J., D'HARCOURT, J., FOLCH, A. and BOFILL, J. *J. de chir., Paris*, 55: 385, 1940.
26. THOMPSON, R. J. C. British Encyclopaedia Medical Practice, Vol. 5, p. 440. Butterworth, London.
27. WEBSTER, D. R., WOOLHOUSE, F. M. and JOHNSTON, J. L. Immersion foot. (A preliminary report as yet unpublished.)
28. GREENE, R. *Lancet*, 2: 23, (6171–241), 1941.
29. GREENE, R. *Practitioner*, 148: 883, 1942.

DISCUSSION

GROVER C. PENBERTHY (Detroit, Mich.): Dr. Brownrigg is to be congratulated, I think, on his study of a very unusual group of cases. It is

timely that this subject be presented to this organization, in view of the fact that many may be called into the naval service to handle this type of lesion; and I think we will all agree with Dr. Brownrigg as regards his classification and his conservative treatment which, of course, means supportive treatment from the beginning, early recognition of the lesion that may require more radical surgery, and at the same time conserving as much as possible.

One in civil life has little opportunity to see frostbites such as have been described. However, I am going to present two cases which were treated by Dr. Johnston, a member of this Association, and describe just what was done with these two patients.

The first individual, sixty-five years of age, was admitted to Receiving Hospital about 1 A.M. Three hours prior to admission, he had been drinking, and on his way home met with a snow drift and fell; he could not get up and the policeman eventually found him and brought him to the Receiving Hospital.

Both hands were markedly swollen and there were blisters. Dr. Brownrigg referred to the paravertebral block. I might say that this particular case was treated by the cervicodorsal block after the technic of Ochsner and Gage, and it apparently did something to add to the comfort of the patient as well as improve the general circulation, as Dr. Johnston described it.

The second case was a gentleman who went fishing out on Lake St. Clair. He wore mittens and felt that his hands were warm, but he started to have a peculiar feeling in his hands, so he returned home. Soon afterward he immersed his hands in warm water, which Dr. Brownrigg has warned against, and was later admitted to the Receiving Hospital. This individual also received multiple cervicodorsal injections.

Both cases eventually required amputation of the fingers.

FRASER B. GURD (Montreal, Canada): I am going to get all the credit that I possibly can from this meeting and draw attention to the fact that Dr. Brownrigg was an ex-resident of mine at Montreal General Hospital, and I knew that he had had this rather unique experience with these unfortunate persons.

Another reason may be that perhaps Dr. Marble's reading may have influenced him, because it is a fact, as some of you may have known, that on more than one occasion I have made contributions (or at least so I thought them to be) on the subject of acute bone atrophy, with regard to the late results of these cases, particularly of trench-foot as we saw it in the last war and in a large number of cases following the last war. I feel, myself, that the late phenomena shown by these cases are tied up with the subject of acute bone atrophy.

Of those in the room, Dr. Donald Gordon, Dr. Herrmann and myself have all written on this subject. We are all at the same level with regard to it in that none of us knows anything about it. I shall not attempt to open

the subject of acute bone atrophy or the late results of trench-foot, because that is not what we are discussing.

SURGEON LIEUT. COMMANDER F. M. WOOLHOUSE, R.C.N.V.R. (Boston, Mass.): I should like to report just very briefly on one very small aspect of the treatment of what we still prefer to call immersion foot, because Lieutenant Commander J. C. White, of the United States Navy, who saw our cases and worked with us on them for over a week, has just reported having seen another large group of cases who were picked up in the Gulf Stream off our southern seaboard. These patients show almost identical lesions, and in that case, of course, there is no question of true frostbite.

We had a total of 142 cases who showed lesions similar to those shown by the cases of Dr. Brownrigg, and in view of the explanation of Freeman, that developing gangrene is simply due to the discrepancy between the local tissue demands and the available blood supply, and that the available blood supply could be utilized by decreasing the local tissue demands, it occurred to us that the simplest and most expeditious manner of doing this would be to apply dry refrigeration. We had no other means at our disposal except the use of ice bags, and after wrapping the feet in sterile towels, after careful aseptic and antiseptic preparation of the feet, the feet were elevated and around the sterile towels were placed several ice bags. Our aim was to reduce the temperature of the feet to about 6°c. below the normal skin temperature.

We found pitting edema, great extravasation of blood, and the feet were often bullatious up to three or four inches above the ankles. We found this difficult to do because of the intense reaction to tissue injury. However, the ice bags were changed at intervals from four hours down to twenty minutes, depending upon the demands of the individual case, and we were able shortly to drop the temperature down.

This produced very dramatic early results. In a period of four hours or less, the edema has practically subsided in the mild cases; in a period of one or two days it had subsided in the serious cases and the skin was wrinkled, and these large blebs, many of them bigger than the palm of one's hand that Dr. Brownrigg described, had resorbed without any break in the over-lying tissue.

One other dramatic result that was shown was that this was not due to elevation alone. We were running skin temperatures on these people with a dermatherm, and in going from one end of the ward to the other, which took approximately half an hour, a man in the first bed, whose ice bags we had removed, and who had had a bleb on the plantar surface of one foot larger than the palm of one's hand, and which had resorbed under treatment with the ice bags, was left exposed in the elevated position. By the time we got to the far end of the ward, all his edema had returned and the bleb was back to its original size.

I have not seen any of these cases for about two and a half months and cannot report on the late results. Dr. Webster will be able to do that. We

did note some early delayed effects. In the first place, a lot of people asked us why we did not employ methods of sympathectomy or sympathetic block, such as had been advised by the French writers during this present war, but which had given extremely bad results. These limbs are all essentially sympathectomized to begin with. Lieutenant Commander White had found that the vessels in the affected limb were sensitized to adrenalin about the end of the ten-day period, as he has reported previously on limbs sympathectomized for other reasons.

We believe that by this method we can save some tissue. How much, we are not quite sure yet.

Among the other early delayed results are intense pain developing about the eighth to tenth day, and in severe cases no subsiding for three or four days. This is a burning sensation with superimposed severe lancinating pain in the exposed extremities. Other effects were the systemic toxemias that Dr. Brownrigg has reported on. Some of our cases showed a transient hematuria and albuminuria, with enlargement of the liver, and one other patient died with a staphylococcal septicemia following empyema.

Another important point that was noted was that one officer with very severe damage to both feet, which Dr. Brownrigg would have classed as a Group II injury, gave a history suggestive of having had a previous renal syndrome, and he was consequently heparinized. We believe this was responsible for saving considerable of his tissue.

SURGEON LIEUT. COMMANDER D. R. WEBSTER, R.C.N.V.R. (Boston, Mass.): These were extremely fascinating cases to work with, and in the main I think we agree completely with Dr. Brownrigg, except that we cling to our immersion feet term.

I have returned from one of the southern Marine Hospitals where I saw some of these cases that had been adrift in the Gulf Stream where the temperature of the water was between 60 and 70 degrees, and two of these patients already were showing gangrene of the feet.

The individual variation in these cases is peculiar. Some of these men in the same boat, and apparently very similar types of men, will go on to gangrene, and some seem to have nothing at all the matter with them, and it seems difficult to predict the moment they come in which are going to recover and which are not.

I have a few slides* and kodachromes of these cases when they came in, showing the initial stages. I am sorry we have not the follow-ups.

I saw these 142 cases that came in about a week ago. Two patients had lost several toes; one patient had had one foot amputated, and there are two more that should be about ready.

Another interesting thing about the patients in the hospital at the present time, who are probably going to lose limbs, is that they are all Australians. They worked in the engine room; they had never before been exposed to cold. Of the several cases I saw from the southern waters, they

* Dr. Webster was unable to show his slides because of lighting conditions.

were all colored but one, which again leaves a contrast that is hard to explain.

LIEUT. COMMANDER THOMAS PETERSEN* (Boston, Mass.): The first boy was in the water only fourteen hours. He still has no sensation in his feet, and he still has rather marked atrophy of his hands.

The temperature of the water that day was 28 degrees and the temperature of the air was 27 degrees. This boy had ulcers of both hands and ulcers of both feet, with bleb formation, and he had marked stiffness of his hands at the time. Now he still feels cold severely.

The second boy came from Florida and has lived in Florida all his life. He was in the water only half an hour, but he walked in the snow twenty-five hours without his shoes. He still has no sensation in his feet at all. He had bleb formation on the heels and on the ball of the foot, all of which eventually subsided, but we still are having quite a problem getting his feet back into condition. He said he felt the cold very badly. His hands are all right.

LOUIS G. HERRMANN (Cincinnati, Ohio): Dr. Brownrigg has brought up some most interesting problems in the study of these cases that I think have taught us a great deal. They have worked out the so-called end or late results. I should like to say just a few words about what we might term the early results. In the Ohio Valley, we have a great number of frostbitten extremities because our natives are not protected very well against cold. They get inebriated, fall by the wayside, and the next morning they have frostbitten extremities.

In the last two years we have seen fifty-seven frostbitten extremities. They are not "immersion" this time, but they might very well be the same physiological problem. We have looked upon these as difficulties falling in the realm of pathologic physiology rather than pathology, and I should like to detail what we might consider the pathologic physiology to cold.

In the first place, cold causes constriction of the arterioles in the extremities and if the cold is not too intense and too prolonged, it is followed by hyperactive hyperemia. If the cold continues, the so-called hyperactive hyperemias which are out in the extremity are in a dependent condition where a great deal of blood is trapped in the extremity, a great deal of venous dilitation or capillary paralysis takes place, and the blood remains fluid in these trapped capillary beds for a long period of time.

In the first stage of this, the ischemic stage, the extremity can bear ischemia for a long period of time without undergoing serious changes. It cannot stand anoxemia or anoxia that comes from the capillary stasis, so before very long, diapedesis takes place, blood cells get out in the tissue and you get that particular slate-gray color of the extremity with or without bleb formation; and we believe very definitely that if the therapeutic efforts can be directed toward the prevention of this particular cycle of pathologic

* Dr. Petersen presented two patients who were in wrecks in February, 1942.

physiology, we will be able to prevent many of these serious complications which we cannot cure by any method we know.

In our experience in this particular type of lesion that we see, the numbness of the foot is the result of a chronic anoxemia of the peripheral nerves. It is an ischemic neurotic, if we dare call it that. We see it a great deal, or did see it a great deal until we became very much impressed by the early signs. We treat these people from the very earliest moment we can get them, and they are kept cold, either by the use of ice in ice bags or any method of refrigeration to keep the demand for oxygen down to its lowest level. That, I think, is very important. The next most important thing, I believe—and I think it is borne out by our own cases, which are nothing to compare with Dr. Brownrigg's excellent series—is that if you can keep the blood circulating by any means, by very gentle superficial massage or by perivascular exercise, these serious complications do not result nearly as often.

We have not lost any extremities in the last five years by amputation. We have lost toes. Our degree of frostbite or frozen extremity is nothing like Dr. Brownrigg's presentation, and I am only telling you this because I do believe more careful attention to the early pathologic physiologic signs is extremely important in the prevention of serious complications from the exposure to cold.

EDGAR L. GILCREEST (San Francisco, Calif.): I think an explanation is due you that I, who spent the first half of my life deep down in the heart of Texas, and the latter half looking for a more salubrious climate in California, should discuss the subject of frostbite, but I saw in the Turko-Balkan war in 1913 (the first war where they really had trench warfare) literally hundreds of these Serbian soldiers, who had stood in the trenches in the snow for hours, with what we called trench frostbite. In Britain in 1915, I saw many who had stood for hours in wet trenches with what we called trench feet.

In the last war I happened to be Chairman of the First Corps of Disability of the A. E. F., where hundreds of these soldiers passed through my hands to be returned to the United States.

Previous to our going into the war, we had for several years heard the argentiferous-tongued William Jennings Bryan say we could raise a million men overnight and we should not get particularly exercised over the European war. We did raise a million men overnight, and they were men, not soldiers. The Negroes were sent out of the Deep South to Hoboken, with shoes that were too tight on their feet. They went down into the bottom of the boat. They could not fish and they did the only other thing they knew how to do, slept and shot craps, and they did not need shoes for that. They got to France. It was January. They put on the too-tight shoes and were put on the train, and they rode in trains that had no heat whatever, and their feet began to swell. Dr. Herrmann mentioned to you the

phenomenon that takes place, and the swelling of the feet in those too-tight shoes.

They were taken off the train, put in an ambulance, never walked a foot of soil in France, were brought to the hospital suffering with these various phenomena of vasomotor conditions, and they suffered greatly. They were brought back to the boats weeks or months later in ambulances and brought back to this country. That cost us literally millions of dollars, but the fact is that we saw all these things, whether you call them frostbite, immersion feet or trench feet. The important thing is that in this war we are going to see a great many of the same things; and if we can do anything now to prevent it, it will be a very important thing.

I think we will have no paper during this whole meeting that will be more important than the ones that we have had this morning. In the case of one man, we had to amputate ten of his toes, and walking with your toes gone is like walking on stilts. I think a paper that will bring out what we can do, is very timely; we should educate the public as well as the army general as to what should be done. The whole thing is a vasomotor phenomenon. A great deal of it can be prevented, and I think anything that will bring that out at this time has been a very important contribution.

WILLIAM DARRACH (New York, N. Y.): I shall take one minute and thank the Program Committee for going not only to Boston and the big cities, but when they want something real for going to Newfoundland. Therefore, on the part of the organization I want to thank Dr. Brownrigg for his delightful presentation and for his long trip down here.

MIMS GAGE (New Orleans, La.): I certainly enjoyed Dr. Brownrigg's paper. Of course, I am down in the South where we do not have very much of this. It is rather interesting to note that we do have it in the Gulf Stream.

I wanted to say a few words about sympathetic block. Dr. Penberthy brought that up, and Dr. Herrmann mentioned the pathologic physiology or the physiologic pathology, and I think in these cases, in the circulation in the foot or extremity, one is dependent upon the other, that is, the arterial, the venous and the lymphatics. As Dr. Herrmann says, once the congestion of the capillaries occurs, the lymphatic flow practically ceases.

Dr. Penberthy showed the excellent results they have from stellate ganglion block, which is so simple that our second and third-year residents do it. It is a simple procedure, and especially in the exudative stage I think it is most enabling, because I have injected about twenty cases of bad infections of the hand in which they had become edematous. We have blocked the stellate ganglion daily for four to five or ten injections, which is very simple and does not produce any damage. You should be careful not to get into the vertebral artery or the dome of the pleura. After the injection in these cases of infected hands which are edematous and sloughish, the patients ask for it because of the relief of pain; and to me the disappearance of the edema in these infections and the rapid healing of the hand is most

dramatic, so much so that I have been afraid to publish it, and I have been doing it for the last six years. I have not published anything because it was quite dramatic and I wanted to wait for a larger series.

I showed Dr. Robert Payne the results and I had a letter telling me that in these infections of the hand with marked edema, and so forth, he had never used anything that gave him more gratifying results; and I think in the exudative stage, in which there is practically complete cessation of capillary pulsation and stoppage of lymph flow, as we have shown in the experimental laboratory, by simple sympathetic block all of this is overcome and the edema rapidly subsides, very much as it does in the thrombophlebitis. For instance, I have seen a case of a very extensive thrombophlebitis of the leg, with the limb swollen from the thigh down to the foot, when it seemed almost as though the skin would rupture from the edema, in which five injections over a period of six days, lumbar sympathetic block with novocaine, brought a normal temperature on the tenth day.

I am sure what others suggested about the application of cold in the first stage is correct, but in the exudative stage I would suggest they use sympathetic block, and I think the results will be most gratifying.

This paper has been of great interest to me. I enjoyed it and I would certainly like to congratulate the essayist.

GARRETT M. BROWNRIGG (closing): I do not think there is very much to add except that I would like to say, in the case of the Gulf Stream, that 50°F. water is still 48° colder than the human body is accustomed to.

HEMATOMAS: EXTRADURAL, SUBDURAL AND SUBCORTICAL

JOSEPH E. J. KING, M.D.

Diplomate of the American Board of Surgery; Director, Neurosurgery, Bellevue, Lenox Hill and Polyclinic Hospitals

NEW YORK, NEW YORK

IN this war period a considerable number of neurosurgeons have entered or will enter the various armed forces. Therefore, general surgeons interested in the surgery of trauma will necessarily have to care for many more cases of craniocerebral injuries.

With this in mind I have prepared the following paper in the hope that it may be of some value to this group of surgeons.

Intracranial operative procedures or explorations are carried out more frequently for blood clots than for any other group of craniocerebral injuries, with the exception of compound and depressed fractures of the skull. In fact, these two groups account for practically all intracranial operations associated with trauma. More often the operation is carried out for a single lesion, but all three varieties of hematoma may be present in the same patient, and they all may be found on one side, one superimposed upon the other. Each group will be considered separately.

EXTRADURAL HEMATOMA

Extradural hematoma is located between the dura and the inner table of the skull. It results most frequently from injury and tearing of the middle meningeal artery, or one or more of its branches. The artery enters the cranial cavity through the foramen spinosum, situated in the base of the skull rather deep in the middle fossa. It passes upward and outward for a short variable distance and divides into two main branches, the anterior and posterior, which supply blood to the major portion of the dura. Tearing through or into this vessel or its branches results in formation of a blood clot between the dura and the inner table of the skull. The size and rapidity of formation of the clot depend upon the site of injury to the vessel and the age of the individual. This lesion occurs more frequently in young adults and patients of middle age, although it may be present at any

age. It is more common in males than in females. Especially in the first decade, the dura is loosely attached to the inner table, and the middle meningeal artery and its branches lie in very shallow grooves, so that an injury to the skull would be less likely to injure the vessel. In the aged group, the dura is more likely to be intimately adherent to the inner table of the skull so that even though a linear fracture might injure the middle meningeal artery or its branches, separation of the dura from the skull is less likely to occur, hence, the paucity of instances of accumulation of blood clot between dura and skull in this group.

A linear fracture of the skull traversing the course of the middle meningeal artery or one or more of its branches is the commonest cause of extradural hematoma, although this lesion also results from more complicated fracture lines including a depressed fragment. In younger adults the fracture line may lacerate some of the smaller branches which do not groove the inner table so decidedly, with the result that the blood clot may be smaller and slower in forming. Doubtless, many instances of small extradural blood clots occur and go unnoticed, because they produce no recognizable signs or symptoms.

The posterior branch is more frequently injured than any other portion of the middle meningeal artery, and occurs as often on one side as the other. The resulting blood clot is biconvex and discoid in shape, similar in shape to the crystallin lens. The size depends upon the amount of injury to the vessel and the ease with which the dura is separated from the inner table of the skull. If the vessel is small and the dura is more firmly fixed, the clot forms more slowly resulting in delay of appearance of the typical signs and symptoms associated with this lesion. When the vessel is completely torn through, with quick outpouring of blood, and when the dura is loosely attached, the clot is larger, is formed more rapidly, and results in death due to the rapid gross compression of the brain in its non-elastic bony case, if the condition is not promptly relieved.

What is stated above refers also to the anterior branch of the artery. The main trunk of the vessel is less frequently injured. One must remember that the trunk of the vessel lies deep within the middle fossa beneath the sphenoidal lobe and nearer to more vital basilar structures of the brain. In this location the clot is more likely to be ovoid, pressing upward beneath the sphenoidal and frontal lobes; and when the vessel is injured at this site, the classical picture is less likely to ensue, due to the absence of hemiparesis or paralysis,

unless the clot rapidly dissects the dura away from the inner table of the skull and extends upward over the motor area.

Extradural hematoma may, but seldom does result from injury of one or more of the larger venous sinuses of the dura, the lateral sinus being more often involved.

Symptoms. The typical syndrome associated with extradural hematoma is pathognomonic. Munro stated, with reference to extradural hematomas: "These are the classical examples and have served as the models upon which the symptomatology of all brain injuries was based."

For practical purposes, all of these clots which come to the attention of the surgeon might be considered acute. I have never had occasion to operate upon a chronic extradural hematoma, unless one of seven or eight days' duration should be so considered. The typical picture is as follows: The patient receives a blow on the head, more frequently on the side than fore and aft, of sufficient force to produce a linear fracture of the skull, more often simple than compounded. Unconsciousness develops, and after a variable interval consciousness returns. The patient may be somewhat hazy and there is usually an interval, the so-called "lucid interval," in which the patient is in control of his senses. He may even telephone from the corner store or some other place, to his family, stating that he has had an accident but feels all right. Following this lucid interval, which may vary from minutes to hours, unconsciousness, perhaps accompanied by hemiparesis or paralysis occurs again, followed by deep coma and deep sterterous breathing. The patient never recovers unless the blood clot is promptly evacuated and bleeding controlled. The pulse is slow and full.

Unfortunately, such a simple picture does not always present itself. Indeed, the symptomatology and diagnosis may be quite obscure. The hemiparesis or paralysis associated with extradural blood clot, when present, is contralateral, and is due to pressure over the motor area. Should the blood clot be low in its position, for example in the middle fossa, without dissecting over the motor area, paresis or paralysis of the opposite side may not be present.

Kennedy and Wortis stated that the presence of a dilated pupil indicates that the clot is on the same side as the dilated pupil. The clot may be bilateral, but this is a rare occurrence. Extradural clot may occur, but seldom does, without fracture.

There may be an underlying homolateral subdural and, or, subcortical blood clot; or an associated contralateral subdural hema-

toma, with or without contrecoup brain damage. The spinal fluid may be clear, pink, or grossly bloody, depending upon the amount of brain injury. Theoretically, the fluid should be clear, but actually this does not always obtain, for the reason that brain laceration in addition to simple compression is often present.

Pathology. The blood clot may vary in size, shape, thickness, color and extent. Its rapid production followed by the acute depression and compression of the brain like a foreign body produces the rapid oncoming symptoms. The hematoma usually consists of solid clots which resemble currant jelly, or they may be as dark as raspberry jelly. Usually the clot is friable, and ordinarily a portion of it is immediately extruded through the burr hole in the skull. According to Vance in his autopsy series, the clot varies in weight from 40 Gm. to 300 Gm., and it may be an inch or more in thickness.

Extradural hematoma complicates craniocerebral injury in about 2 to 5 per cent, and probably is less common than is generally believed. Vance's figures show that a considerable number of cases are never recognized except at autopsy. There were 106 instances of extradural hematoma in his series of 507 autopsy cases, of which sixty-one clots were of sufficient size to produce death through cerebral compression. He stated that the duration of the clinical course was variable, and that over half of the patients died within twenty-four hours. A large proportion were found dead. He advanced the belief that a number, after injury and during lucid intervals, wandered away from the scene of the accident, possibly in fairly good condition, and were overcome by the rapid on-coming stupor and coma, and died without observation. Others were found in deep coma so profound and so prolonged that operation was of no avail. No doubt, in many the condition was confused with the alcoholic state and was not recognized.

Treatment. From the foregoing one can judge that the treatment is surgical demanding immediate removal of the blood clot. If operation is too long delayed, compression of the brain stem takes place to such an extent that the condition is irretrievable. Immediate operation is probably never indicated in cases of craniocerebral injury so positively as in the case of extradural hemorrhage. The urgency of operation is almost as great in some instances as in stab wounds of the heart. In my own experience the presence of the surgeon in the hospital at the time of admission of the patient prevented the loss of life in more than one instance. Had it been necessary to call the surgeon from any distance the period of safety would have passed.

If time permits, the patient should be taken to the x-ray room on the way to the operating room so that films, especially lateral films, may be made. In view of the fact that a large percentage of these is associated with linear fractures of the skull, the position of the fracture line, in addition to other clinical evidence, may indicate the point at which the burr hole should first be made. The films can be developed and viewed as wet films while the head is being shaved and the scalp prepared.

Operation is carried out under a local anesthetic. In some instances the stupor or coma may be so deep that no anesthetic will be needed until after evacuation of the clot. The patient may then quickly regain consciousness, and local anesthesia may be required for proper closure of the incision.

The burr hole is made over the site selected through a muscle-splitting vertical or oblique incision, depending upon the position of the clot. The burr hole may have to be made over the postparietal or in the temporal region, and in instances in which one is led to believe the clot is low in position and probably in the middle fossa, the opening should be made low in the squamous portion of the temporal bone just above the zygoma. Osteoplastic flap is mentioned only to be condemned. No time should be wasted in application of numerous artery forceps and skin clips to the scalp. Self-retaining retractors will suffice to provide adequate exposure and control bleeding. When the burr hole is made, a considerable amount of clot will be spontaneously extruded through the opening, if it is properly placed. It is advised that this opening be enlarged so that proper evacuation of the clot and control of hemorrhage may be accomplished without tedious delay, especially when the clot is rather large and thick and in the temporal region. The musculature and scalp will give adequate protection. In instances in which it is not necessary to open the dura, or in which the dura has been opened and closed, and is approximated to the pericranium, the bone will re-form anyhow. In a number of instances evacuation of the clot will suffice. This is done by suction and curettage will not be necessary. The bleeding may have stopped, but if it is mild the bleeding points can readily be controlled by suture ligatures or electrocoagulation.

When the fracture line involves the trunk of the middle meningeal deep in the fossa, the active bleeding may be considerable, and the site of the injury to the vessel may be found with difficulty, if at all. It may be controlled by narrow gauze packing brought out through the lower angle of the incision and removed gradually over a period

of four or five days. Should there be any doubt as to adequate control of bleeding, it is advised that immediate ligation of the external carotid artery be done as indicated in some cases of nose bleed. Ligation will rarely be indicated. The ligation can be done in a few minutes and will add but little risk. Collateral circulation will not prove troublesome.

Should the dura appear "bluish" it should be opened and the subdural clot removed. It is advised that the dura be loosely closed leaving an interval between sutures for the escape of any subdural fluid which might accumulate.

Munro advises opening the dura in all cases, but I see no necessity for this except in the presence of clot or free fluid. With this Coleman agrees. Creation of a dural opening for decompressive effect is seldom indicated. Should a subdural clot be found and removed, a small rubber dam drain is inserted just beneath the dura between sutures, and removed after twenty-four to thirty-six hours. After complete removal of all clots and loose closure of the dura, it is well to fix the elevated dura to the overlying pericranium with sutures in order to obliterate the "dead space" which would otherwise exist. The incision is then closed in layers with interrupted silk sutures. Blood transfusion and infusions may be indicated. In one instance I observed the three types of blood clots through the same incision, the one superimposed on the other, and after removal of all three clots, the patient recovered.

Prognosis. In uncomplicated cases, rapid and complete recovery may be expected. The patient may "wake up" on the table and have to be restrained by assistants and belts. On the other hand, the tremendous degree of compression may be so great and prolonged that the patient will not recover regardless of the adequacy of the operation. One must always consider the possibility of a concurrent injury or lesion, and should bear in mind that the blood clot may be bilateral, especially in circumferential linear fractures. There may be a subdural clot on the opposite side, or gross damage by contrecoup. If the patient's condition does not show the expected improvement following evacuation of the extradural clot, one should not hesitate to make a burr hole on the opposite side. It is humiliating to see the patient lapse into coma followed by death, and have autopsy reveal a large clot on the opposite side. It is even more humiliating at autopsy to find that the burr hole was placed a short distance from the margin of the blood clot without localizing it. Probably most of us have had this experience.

In conclusion, practically all patients with uncomplicated extradural blood clots overlying the cerebrum should promptly recover if the operation is performed at the proper time, the blood clot is completely removed, and the bleeding adequately controlled. Unfortunately, many cases are seen too late, and others show severe complications.

With reference to the extradural clot of the posterior fossa, a few words might be said. Here the clot most usually results from injury of the lateral sinus or one of its tributaries, although it could be arterial in origin. Smaller clots may prove to be more quickly disastrous on account of the small space involved in the posterior fossa, and the fact that the compression involves the more vital centers. Fortunately, this group is very small. It goes without saying that evacuation of the clot and control of bleeding should be carried out immediately, and even so the prognosis is not favorable.

SUBDURAL HEMATOMA

Although the condition was described by Virchow, in 1857, and termed "pachymeningitis hemorrhagica interna," and four typical chronic cases were reported in their entirety by Trotter, in 1914, subdural hematoma as an entity was not properly recognized until after the report of Putnam and Cushing, in 1925. When one considers the fact that the lesion frequently results from the mildest kind of trauma or a bump on the head, one must concede that this lesion most assuredly has occurred since the beginning of mankind. It is almost unbelievable with the other advances in modern surgery, that this condition evaded recognition by the medical profession until recent times. Only a few articles on this subject appeared in the literature in the late 1920's after the paper by Putnam and Cushing. However, since 1930 many splendid articles and papers have made their appearance. Now, the "typical" subdural hematoma is recognized by neurologists and neurosurgeons as an entity, just as is the extradural clot.

The occurrence of this lesion seems to be frequent in recent times contrasted with its almost complete lack of recognition prior to 1925. During our intern days in 1909 to 1915 we heard of the diagnosis of "pachymeningitis hemorrhagica interna" made at autopsy by Doctors Charles Norris and Otto Schultz, but further than the diagnosis the condition was not understood. It was not known as a "subdural blood clot." We were completely ignorant of the nature of

this lesion at that time. Deaths resulting from this type of clot evidently were ascribed to a "cerebral attack," apoplexy, or some other intracranial episode which was not understood or appreciated. Even in this day and time, most cases of chronic subdural hematoma are referred by the patient's physician to the neurologist or neurosurgeon with the probable diagnosis of "brain tumor."

General surgeons who may be most proficient in all their other undertakings, may not be sufficiently aware of the presence of a subdural clot which could readily be evacuated by any one of them, were the condition recognized. Considering the fact that these accidents occur over such a widespread area, and the paucity of neurosurgeons, it behooves the general surgeon, especially those who devote much of their time to traumatic surgery, to pay more attention to the trivial accidents that result in this condition which may be so readily relieved by simple trephine in many instances. For this reason, emphasis on this subject is made.

Subdural hematoma should be considered as "acute" or "chronic" hematoma.

Acute Subdural Hematoma. It is well understood that if an acute hematoma is not removed, and if the patient lives sufficiently long, the lesion becomes "chronic"; that is, the chronic lesion is merely a later stage of the acute condition. No time limit has been set to indicate the termination of the acute stage and beginning of the chronic stage. Kennedy and Wortis, in their paper, included in the acute group only cases occurring within twenty-one days.

From the practical standpoint, acute cases should be subdivided into two main groups: First, those associated with massive and gross trauma of the skull and brain, in which the subdural clot may or does play a minor rôle; and second, those cases which usually follow minor trauma to the head, with no injury to the skull, and but little injury to the brain and its coverings. In the second group the subdural clot plays a major rôle.

In the first group most of the patients will die with or without treatment. Even when the clot is removed the mortality is very high. Consideration of the treatment and management of this group does not fall within the scope of this paper.

In the second group it is believed that a "simple" uncomplicated acute subdural blood clot results in most instances from a mild blow, probably on the anterior or posterior aspect or pole of the head. The blow or trauma may be so mild that it will not be remembered by the patient, or the patient when examined may be in such deep stupor or

coma that no history of a blow can be obtained. The blow may be so trivial that no evidence of it can be found by examining the scalp, and it is usually not associated with fracture of the skull. Most writers believe that the clot forms as a result of tearing or rupture of the cortical veins in the free subdural space after their coalescence and before their entrance to the major or venous sinuses of the dura. It is believed that the trunks of the cortical veins suspended in the subdural space between the cortex and the dura are torn through by propulsion of the brain bulk forward or backward, after sudden arrest of motion of the skull by the impeding object against which the skull is struck. In most instances in which history can be obtained it will be determined that the "head was struck against something" rather than the "head was struck *by* something"; i.e., the head was in motion. Typical examples are cases in which the patient stoops over or leans backward and strikes the head against a table, piano, bathtub, etc. In all these instances the motion of the head is suddenly arrested, while that of the brain continues. The force and excursion are sufficient to tear the cortical vein or trunks, without necessary contrecoup damage to the poles of the brain. One might crudely but graphically compare this picture with that of a person standing in a wagon-bed, driving a team of horses at full tilt. Should the horses stop suddenly and unexpectely, the driver most likely would be propelled forward against the wagon-bed. The shift of the brain in its case is greater in the fore and aft directions than from side-to-side, due to the fact that the falx cerebri acts as a stabilizer in the lateral direction, as well as the fact that the brain bulk is greater in the anteroposterior diameter. The greater width and fixation of the falx at its posterior extremity may account for the fact that the clot usually extends more over the frontal than the occipital pole of the cerebrum.

In addition to the assigned type of trauma, there must certainly also be some predisposing cause, such as short trunks of the cortical veins, weakness and friability of the venous wall, etc. No doubt every living person has struck his head, not once but many times, with various degrees of violence, yet only a few persons suffer from formation of a subdural clot. In fact, a clot may form from spontaneous rupture of a vein in the subdural space without evident trauma, a fact also recognized by Virchow. Halpern reported a case in which a cerebral aneurysm ruptured at the point where the internal carotid passes through the subdural into the subarachnoid space to continue as the left middle cerebral artery. The author observed the same

condition in a patient about four months ago. In this patient the pupil alternated several times between normal size and marked dilatation. The aneurysm was no larger than a split pea.

A large subdural clot may also result from injury of the major venous sinuses and may terminate in early death. It may also be the result of complete division of the middle meningeal artery with a markedly adherent, lacerated dura. The proximal end of the artery may project through the rent in the dura and quickly pour its full volume of blood into the subdural space. If it is not removed immediately, death promptly ensues.

When the vein is torn, free bleeding takes place over the cortex, usually the cerebral, with varying degrees of rapidity and volume. Should the anterior veins beneath the frontal or sphenoidal poles be torn, the clot may extend downward beneath these lobes, or upward and backward over them, or both. Or, the injured vein may be posterior in position, with the major portion of the clot in this vicinity. More commonly one finds the clot extending over the cortex occupying practically the entire length of the cerebrum from pole to pole, in an anteroposterior direction, and from near the midline downward below the upper portion of the squama. Therefore, its horizontal length is usually greater than the vertical, and thus differs from the discoid or ovoid form of extradural clots. The clot is thickest in its central portion, and biconvex in shape, the external surface being greater in convexity. It may be an inch or more in thickness. The clot may be solid, having the typical dark blue-black color of clotted venous blood if seen early after its formation, if only venous damage has occurred. Frequently, the pia-arachnoid will have been torn, in which instance the clot will be more fluid in character. In fact, the bulk of the mass consists of dark bloody fluid, decidedly more fluid than clot, and can be readily evacuated through a small incision in the dura.

The cerebral mass is displaced downward and toward the opposite side by the clot in unilateral lesions; if bilateral, downward only for the most part. The associated symptomatology is due to the compression and displacement of the cerebrum rather than to the internal loss of blood *per se*.

The pathology, including composition, organization and partial liquefaction of the clots has been fully and adequately described by Munro and Merritt, Putnam, Gardner, Zollinger, and other writers, and will not be considered in this paper, primarily intended for the practical use of the surgeon.

Symptoms and Signs. One could expect that a displacing lesion of such large proportions would be followed by bizarre signs and symptoms. This may or may not be the case. The lack of signs and symptoms may be marked. There may be only mild headache. Should this increase, a physician may be consulted, and after a cursory examination the patient may be dismissed with a prescription for medication for the relief of headache.

This picture in cases of simple uncomplicated acute hematoma may prevail from hours to several days. Sooner or later, however, certain signs and symptoms will appear, partly subside or disappear, and then recur. Although there is no pathognomonic syndrome, as is present in a typical case of extradural hematoma, it is believed that the effect on consciousness in the "in and out" picture, as we call it, is one of the most constant findings and leads one to suspect promptly the presence of a subdural clot. More often one may obtain the history of some injury to the head from the patient, family or witnesses. There may be a period of loss of consciousness from two to twenty-four hours, headaches, and irritability, from all of which he may apparently recover, with a lucid interval of three days to several weeks or months. On the other hand, drowziness may progress into deep stupor and coma, from which the patient may not recover.

All varieties of changes in the reflexes and peripheral pareses or paralyses have been observed. Unilateral central type facial weakness is common. The hemiparesis or paralysis, when present, may be home- or contralateral, possibly about equally divided, but in my experience, more often on the homolateral side. To my chagrin, in the earlier days before this fact was known to me, I had a disappointment similar to that of Grant, Gross and others, in that only one exploratory burr hole was made, and that on the contralateral side. This patient had been walking about for five days after a mild blow on his head, and came to the hospital under his own power. At autopsy an enormous clot was found on the homolateral side.

The pupil may be dilated and fixed without ptosis of the eyelid. Some writers state that the dilated pupil is found as often on one side as the other, with reference to the location of the clot. At Bellevue Hospital we noted that it was more common on the homolateral side, and was one of the chief indications for selection of the site of the first burr hole.

The cerebrospinal fluid may be clear and of normal contents, if there is no laceration of the brain, but may be xanthochromic, pink

or grossly bloody depending upon the amount and position of the brain damage or laceration. It usually shows considerable elevation of pressure by the manometer in this stage, but the pressure may be within normal range. The pulse may be slow, normal or increased in rate, and may vary in rate and volume even while the hand is on the pulse. Leukocytosis is usually observed due to the presence of the blood clot or blood in the cerebrospinal fluid or both. The temperature is not markedly elevated in the simple uncomplicated cases. Should it be above 103°F., it is due most likely to a concomitant infection or severe brain damage, and the outlook is poor. Papilledema is not commonly found in the early stage, but engorgement of the retinal veins may be observed. At the time of the examination in the hospital the patient may be conscious, stuporous or in deep coma.

Diagnosis. The diagnosis may not be made readily on admission. Dehydration therapy is usually carried out in the time consumed in making the diagnosis. One should strongly suspect the presence of an uncomplicated acute subdural hematoma with the following findings: history of slight injury to the forehead or back of head followed by a period of unconsciousness lasting from a few minutes to hours; stupor, or loss of consciousness, especially of the "in and out" type; a lucid interval of several hours or days; negative x-ray films of the skull; hemiparesis; dilatation and fixation of one pupil, with lumbar puncture revealing cerebrospinal fluid clear, xanthochromic or pink, and usually under increased pressure. Other signs and symptoms may be of additional value, but those enumerated constitute sufficient evidence to warrant strong suspicion of the presence of an acute subdural hematoma.

If an electro-encephalographic apparatus is available, and the patient's condition permits, electro-encephalograms may prove of great value, not only in determining the presence, but also the lateralization of the lesion. This coincides with the opinion of Schwab. One might expect that air studies would reveal marked distortion and displacement of the ventricles, but on the whole, air studies have been of little value. Substitution of air by the lumbar route, even in the presence of low or normal manometric pressure, might prove disastrous due to jamming of the conus, or herniation of a part of the cerebrum through the opening in the tentorium. Manometric pressure of the spinal fluid is not always the same as that of the intracranial pressure. Ventriculograms will be precluded in most instances for the reason that the clot will probably be found

when bilateral burr holes are made. Should no clot be located when the holes are made, ventriculograms are then indicated and should be made at this time.

Treatment. Except for supportive treatment in addition to dehydration previous to operation, the most essential thing is removal of the clot. Operative procedure should be carried out under local anesthesia. The head should be placed in position and draped so that bilateral exposure is obtained. In every instance, with no exception, bilateral burr holes should be made, for the reason that the subdural clot is frequently bilateral. Ira Cohen stated that, if the clot is unilateral, he would prefer making the burr hole on the wrong side first, so that if no clot is found, he will be sure to investigate the opposite side. Some prefer the temporal approach, and some the postparietal. Multiple burr holes may be necessary in the localization and removal of the clot. At any rate, the burr hole is made through an incision not more than 1¼ inches long, and it is well to make both openings before the dura is incised. On the side of the clot the dura will appear discolored—blue, or bluish-green. When the dura is incised, a "liquid clot" will evacuate itself without difficulty; in fact, the fluid pours out in varying amounts with pulsation. Some surgeons advise irrigation of the subdural space with saline to facilitate removal of the blood elements. Others advise strongly against it in the belief that it increases edema of the brain. A rubber dam drain should be inserted into the subdural space through the small incision, and brought out through the lower angle of the incision in the scalp, and the scalp closed. The drain is partly removed in twenty-four hours, and completely removed after forty-eight hours. This simple operative procedure suffices with liquid clots.

Should the clot be solid, consisting of typical coagulated blood, it is advised that the opening on the side of the clot be enlarged quickly with rongeurs to the size, approximately, of a silver dollar and the incision in the dura be lengthened. By so doing, the solid clot can be readily and thoroughly removed by suction under direct vision. Passing the suction tip in all directions without direct vision may result in tearing into another vein, with re-formation of the clot. Therefore, this should be avoided. When the clot has been evacuated, it may be observed that the cortex will be depressed, although the convolutions otherwise may appear normal. The surface of the cortex may be stained by the hematogenous elements. The depression in the cortex may disappear immediately. When the depressed cortex becomes vigorously elevated, marked increase of the intraventricular

cavity can be suspected. After complete evacuation of the clot, the dura is loosely closed with a rubber dam drain inserted just within the subdural space. This is removed during the next twenty-four hours.

Munro rightly states that the postoperative care is very important. In all instances we have performed postoperative lumbar punctures, sometimes daily, until the patient is "out of the woods." (Previous to operation a lumbar puncture is done only for diagnostic purposes.) Dehydration may be done, but in the majority of instances will not be found necessary. Reaccumulation of the clot may occur, and one must be on the alert for this possibility. It is not a common occurrence.

Prognosis. In the majority of patients operated upon after the seventh day, recovery should be expected, and is usually without untoward event. The high mortality reported by Kennedy and Wortis, and various other writers, is due to the fact that the first group of cases, mentioned but not considered in this paper, is included. Kennedy and Wortis reported 43.7 per cent in the operative mortality in thirty-two cases from the Neurological Service of Bellevue Hospital. Thirty-one patients died without operation. Other writers report an equal or higher mortality when cases of gross skull and brain damage are included.

Frank Turney recently informed me that the approximate operative mortality rate at Kings County Hospital was as follows: During the first twenty-four hours, 88 per cent; from first to third day, 70 per cent; from third to seventh day, 42 per cent; from seventh to eleventh day, 15 per cent; from eleventh to fourteenth day, between 8 and 10 per cent; and from fourteenth to twenty-first day, 12 per cent.

Francis Echlin informed me that during the approximate eighteen months in which he has been in charge of the neurosurgery in the Bellevue Psychopathic Hospital, they operated upon about eighty patients with subdural hematomas with the following results: Very few were operated upon during the first twenty-four hours. Most of them were operated upon after the third day, especially after the seventh day. Practically all those operated upon in the first twenty-four hours died; while the majority of those operated upon after the fifth day recovered, provided they were not in prolonged, deep coma. From these figures and statements it can be concluded that the majority of patients operated upon should recover, provided there is no associated gross damage of the skull and brain, or deep, prolonged

coma. In the Kings County Hospital series, the slight increase in death rate between the fourteen and twenty-one days was due to the concomitant infections, pulmonary involvement and general debility.

Chronic Subdural Hematoma. If an acute subdural hematoma is not recognized and removed, and the patient survives, it becomes chronic. It may be present for weeks or years without being recognized. The outer and inner membranes may be calcified with a liquid central portion. Lillie reported removal of a completely calcified lesion over twenty years after the injury.

Some patients may show practically no signs or symptoms, and certainly none sufficient to justify operative interference. On the other hand, it is believed that a history will be obtained either from the patient or members of the family which will cause one to suspect the presence of a chronic subdural clot. The symptoms of irritability, lack of interest, inability to work or concentrate will, most likely, be brought out by proper questioning. His friends will have noticed that the patient has changed, that he does not behave normally, and that he acts queerly.

The one outstanding feature in many cases is headache, intermittent, or continuous, sometimes mild, but in many cases very severe and constant, necessitating cessation of work. Nausea and vomiting may accompany the headache. It may be unilateral, but is often generalized, and is of a persistent, throbbing character. Coleman stated that in over half of his series of cases, headache began immediately or within two days after injury. Delusional and maniacal manifestations may be present, and some patients have been confined to institutions for the insane.

History of trivial injury may be obtained. Patients in stupor, who cannot give a coherent history, may recall the injury a few hours or days after evacuation of the clot. There may not be any period of unconsciousness. Stupor or unconsciousness alternating with consciousness is not uncommon, and constitute the "in and out" state so often spoken of by Foster Kennedy. The gradual passing from drowziness to stupor, loss of consciousness deepening into coma, may be observed, and bodes ill unless the clot is immediately removed. This picture is most likely to occur during the first three or four months, but may occur in patients with older clots.

Hemiparesis is present in about 50 per cent of the group, and is more often homolateral in the early cases, and contralateral in the late or very chronic stage. This occurrence is explained by the fact that the lesion in the earlier stage is a fairly rapidly expanding soft

lesion which presses the opposite hemisphere against the skull, flattening the convolutions, or the brain stem against the opposite sharp margin in the incisura in the tentorium, or both. After many months or years the brain is not so likely to be affected in this manner. The clot remains more or less as a simple foreign body, like a tumor, locally compressing and displacing the underlying motor area, thereby producing contralateral motor weakness similar to that of a tumor.

There may be homo- or contralateral dilatation of the pupil, but this is not so frequently seen as in the acute cases. Papilledema may be very marked or there may be no changes in the disk. Most of the cases in this group are referred to the neurologists and neurosurgeons as "brain tumor suspects," and the clot may be found when the burr holes are made for ventriculography in the localization of the suspected tumor. The diagnosis of "allergy of the brain" has been made. Frequently, the positive diagnosis is established after the burr holes are made.

Treatment. As in the case of acute subdural hematoma, the treatment is surgical, and consists of removal of the clot. Local anesthesia is used. In all instances the clot should be removed in its entirety if the condition of the patient will permit. In a few cases in which the condition of the patient is very poor, it is advised that the liquid portion of the clot be removed through a burr hole with no loss of time, as suggested by Coleman, wait for improvement, and remove the clot in its entirety at a later date. It is not believed that chronic organized clots can be successfully removed through a burr hole, nor is it believed advisable that only the liquid portion should be removed, leaving the bulky membrane in position. Bilateral burr holes in the upper temporoparietal region should always be made for the purpose of making a diagnosis, because a number of these lesions are bilateral. The clots may be of equal or unequal size. Burr holes are preferred to air studies in the majority of cases. The lesion most likely will be found and can be removed at the same time. If the clot is of fairly recent formation, so that the outer membrane strips easily from the dura, it can be removed more readily and completely after enlarging the burr hole, as is done for acute subdural hematoma. Coleman prefers this procedure to a bone flap.

In the very chronic clots which have existed for many months or years, the dura may even then appear bluish or greenish-blue when viewed through the burr hole, or it may not be discolored. In the latter, the dura offers a wood-like resistance when cut, and only after

incision of the outer thickened portion of the dura, will the dark color of the inner portion be seen, indicating the true nature of the underlying clot. In such cases the outer membrane will have become so firmly organized and fixed to the dura that it may be removed with difficulty even after a flap has been turned.

This type of clot cannot be properly removed through a simple burr hole, and since the bony opening would have to be enlarged to undesirable proportions, I have preferred making a bone flap through a straight or slightly curved horizontal incision. The incision is made in the direction of the branches of the supra-orbital nerve, which extend far backward toward the occiput, thereby resulting in a smaller, postoperative anesthetic area of the scalp. The galea and scalp are reflected from the pericranium and outer fascia of the temporal muscle exposing an area larger than the palm of the hand. The bone flap, turned downward and hinging on the temporal muscle, is longer in the horizontal than the vertical direction, being in the proportion of about three inches to two inches. The bone flap is so fashioned for the reason that the underlying firm clot is longer in the anteroposterior than in the vertical direction. After the dural flap is made, the outer membrane is incised and the fluid portion of the clot is evacuated by suction. A considerable amount of the inner, soft grumous and friable portion of the clot is also removed by suction. The bulk of the mass is thus decreased, but there is no tendency whatsoever to spontaneous separation of the thick, tough, adherent outer membrane from the dura. With care and a little time the clot can be stripped away from the dura. Sometimes considerable force may be required, comparable to that exerted to strip the lining from a chicken gizzard. The brain spoon is an effectual instrument for this purpose. One should be especially careful in freeing the peripheral margins, otherwise tearing of a cortical vein or the pia-arachnoid and cortex may occur. This portion of the clot is often quite adherent to the pia-arachnoid. Small pieces of clot should be left attached rather than cause damage to the underlying veins and cortex by complete removal. Recurrence of the clot will thus be prevented. Removal of the inner membrane occasions no difficulty due to its very loose attachment.

After removal of the clot the cortex fails to expand immediately in most cases of both acute and chronic subdural hematoma, as stated by Coleman, but contrary to the statements of some writers. I am unable to explain this phenomenon. A rubber dam drain is inserted beneath the dura, as described, and is brought out through

a burr hole at the upper posterior portion of the flap. After loose closure of the dura, the bone flap is replaced and fixed with three rustless steel wire sutures passed through small drill holes. The scalp incision is quickly closed in two layers. The drain is slowly removed and shortened over a period of three or four days, during which time a considerable amount of fluids may be given with the patient in the prone position, to aid in expansion of the brain.

Although the postoperative course may be uneventful, some patients may have a very stormy period, even after a few days of well-being. They may require dehydration. If manometric readings show increase of pressure from 190 to 250, therapeutic lumbar punctures are done, daily if necessary. Curiously, the contralateral hemiparesis may increase several days after removal of the clot, even amounting to hemiparalysis for a variable period. Since the drain is removed over a period of several days, reaccumulation of fluid in the subdural space is not expected. I am not able to give definite information regarding this possibility for the reason that the flap has never been re-elevated. It is believed that the increase in paralysis, persistent for several days or weeks, may be due to dislocation of the motor cortex and the connecting motor fibers, resulting from the expansion and elevation of the depressed cortex. The cortex has been depressed so long that it may have become more or less adjusted to the compression. When this is suddenly relieved, the depression of the cortex should disappear sooner or later. It is quite conceivable that the change in the relationship of the motor area and motor fibers could be as great following expansion as compression of the cortex. In other words, dysfunction of the motor area can be caused by the marked displacement due either to its expansion or compression. Increase in the edema of the cortex probably accompanies the expansion and may account to some degree for the increase in the paralysis. During this period, convulsions involving the paralyzed side are prone to occur.

Prognosis. A good, and frequently complete recovery can be expected in the vast majority of these cases, regardless of the rather stormy periods through which some of them pass. If operation is performed on a patient who has been in deep coma twelve hours or more, the result will probably be fatal. Should coma be of short duration, or only deep stupor, recovery can be expected. Deep coma caused by chronic subdural clots, induces or produces irretrievable changes in the vital centers in a relatively short time compared with that associated with tumor. It is believed to be due to a sudden and

violent increase in intracranial pressure. One may note a similarity to
the mechanics and sequelas associated with gross hemorrhage about
or in a tumor. If the coma develops shortly after admission, opera-
tion will likely prove effective if carried out immediately. Sachs
stated that all patients in his series operated upon in deep coma,
died; all others survived. The other chief causes of death are infec-
tion, pulmonary complications, and general debility, especially in
older people. On the whole, satisfactory results may be expected in
this group of cases. Great satisfaction is experienced in the return of
these patients to their homes and livelihood, most of whom would
eventually die as a result of the clot, be relegated to a life of useless-
ness or become inmates of an insane asylum.

Subdural Sacculated Collections of Fluid. A collection of fluid in
the subdural space has been known by various names, e.g., "fluid or
liquid-hematoma," "hygroma," "hydroma," etc. It seems that one
name is as applicable as another, and probably that "hematoma" is
the least acceptable. "Hematoma" merely means a "blood clot,"
and by no stretch of the imagination can the lesion under considera-
tion be a clot. A clot consists of both solid and liquid portions, but
the solid predominates. I do not consider a subdural collection of
cerebrospinal fluid, originally mixed with a small amount of blood, a
hematoma, anymore than intraventricular or sacculated intraspinal
fluid mixed with a small amount of blood. The fluid may be colorless,
xanthochromic, pink, or somewhat red. Regardless of the terms used,
all writers seem to agree as to the nature, symptoms and signs of the
lesion, and since they are very similar to those of subdural hema-
toma, this lesion is briefly discussed. It is supposed to result from a
tear of the pia-arachnoid, with leakage and collection of cerebro-
spinal fluid in the subdural space. However, one cannot account in
this manner for similar collections of non-infected fluid associated
with otogenic conditions and osteomyelitis of the skull. The propor-
tion of hematogenous elements is small.

The collection of fluid may be unilateral or bilateral, comparable
in size and extent to a subdural clot. The treatment is simple and
consists of removal of the fluid through a burr hole, preferably placed
over the posterior pole of the collection of fluid to favor more com-
plete evacuation in the prone position. A small rubber dam drain is
used and removed after forty-eight hours.

This condition should always be borne in mind in dealing with old
head injuries regardless of whether they are compensation cases or
not. These patients should not be glibly or lightly dismissed, tagged

as "post-traumatic neurosis." This term is so easy to use. My attention was first called to the condition in 1923 before the appearance of the paper by Naffziger. An ex-soldier had received a glancing gunshot wound of the anterior portion of his head several months previous to my examination. The bullet struck the outer table of the skull just within the hair-line, without fracture of the skull. It was not known whether the patient fell and struck his head. The wound healed leaving a small oval bald spot about an inch long, about three-quarters of an inch left of the midline. He complained bitterly of headache and tried to jump out of the window to commit suicide. He had no other symptom or sign on neurological examination. The attending physician called him a "neurotic" and a "nut," and paid no attention to him. Several months after listening to his complaints, and without doing anything for him, the physician prevailed upon me much against my better judgment at the time, to make a "psychologic" burr hole directly over the site of the bald spot. I did so, using local anesthesia. When the dura was incised, an unbelievable amount of clear, colorless fluid poured out through the dural incision. After some of the fluid had escaped, the patient said: "Doctor, my headache is going," and he had no idea as to what I was doing. I increased the size of the burr hole with rongeurs and lengthened the incision in the dura so as to get a peek beneath it. After a great amount of fluid had escaped, I was truly amazed at the considerable capacity of the empty subdural space. The cortex remained depressed and showed very little evidence of becoming elevated. The patient's head was turned to the left side so as to lower the level of the opening, and he was placed in Trendelenburg position. The change in position of the head was followed by escape of much more fluid. After the patient had been returned to the original position, the incredibly commodious subdural space was gazed at. Reasons for the existence of this collection of fluid were not apparent at the time. Upon conclusion of the operation, the patient stated that he had no headache for the first time in months, and that he felt quite well. He made an uneventful recovery, to the surprise and chagrin of the attendants and myself. As a matter of curiosity and interest, he was kept on the ward for a period of at least eight months, during which time he made no complaints. Since this episode I have seen a number of similar cases. It behooves the medical profession to consider well the likelihood or possibility of this lesion before the diagnosis of "post-traumatic syndrome" is made.

Subdural Collections of Fluids in Infants. This condition is usually mistaken for hydrocephalus, brain tumor, some kind of birth injury or congenital abnormality. The head enlarges slowly and gradually, differing from the more rapidly enlarging head in true obstructive hydrocephalus. The veins of the scalp are dilated and prominent. Although the baby may take its feedings, it may refuse them from time to time without apparent cause. It has bouts of fretfulness. Accompanying the slow enlargement of the head, changes of the fundi will be observed. Hemorrhages are not uncommon, and choking of the disks in varying degrees will be present. Convulsions occur and may or may not be associated with hemiparesis. The lesion is frequently bilateral.

The diagnosis can be established by puncture of the subdural space through the lateral recesses of the fontanel. Repeated punctures for therapeutic reasons should not be done because they are ineffective.

Treatment. Munro advises removal of the fluid and a portion of the membrane and states that "it does no harm to leave some of the membrane in place." He and others advocate the subtemporal approach. I prefer making a small flap of bone about 2 inches in diameter, in the temporoparietal region, turned down through a three-inch straight incision similar to that for subtemporal decompression. Local anesthesia is used. The scalp flaps are readily reflected from the pericranium and outer temporal fascia and are held with self-retaining retractors which afford adequate exposure and control of bleeding from the scalp. Two burr holes are made, one anteriorly and the other posteriorly in the upper part of the exposure, about 1½ or 1¾ inches apart. The skull between the burr holes is cut on a bevel with the Gigli saw. It is quite thin and cuts readily. The remainder of the flap is cut with an especially small deVilbiss bone forceps and the flap is hinged below on the temporal muscle. All of this can be done in five or six minutes on account of the thinness of the skull. Bleeding is minimal. A similar, but inverted flap, is made in the dura. When the dural flap is reflected upward, one sees a perfectly transparent corneal-like membrane, with a slight pinkish or flesh-colored tint. One can view the depressed surface of the cortex through the membrane and its enclosed fluid as clearly as one sees the bottom of the ocean through a glass-bottomed boat in the Bahamas. It makes a pretty and interesting picture. The membrane offers a surprising degree of resistance to the puncturing knife, considering the appearance of the cellophane-like structure. When it

is incised, the fluid escapes freely and the sac collapses. The fluid may be colorless or faintly yellow. The membrane is grasped with a forceps and a general drag is made to determine its points of attachment to the pia-arachnoid. The lesion occupies a position similar to that of a subdural hematoma. Most of the membrane can be removed. Force should not be used lest a cortical vein or the pia-arachnoid be torn. A small piece of the membrane left attached here and there does no harm, and laceration of the vein of the cortex should be studiously avoided. After removal of the fluid and the membrane, the depressed cortex does not become elevated, even when a similar lesion is known to exist on the opposite side. In this respect the cortex behaves in a similar manner to that described in the cases of old, chronic subdural hematoma. Therefore, a small Penrose rubber dam drain is fixed between two of the dural sutures along the posterior limb with the distal end protruding about $\frac{1}{4}$ inch beneath the dura into the "dead space." The proximal end is brought out through a small stab wound in the posterior scalp flap and fixed to the scalp with a silk suture to prevent possible displacement. The bone flap is returned to its position and fixed with one rustless steel wire ligature, passed through small drill holes. Thus, no gross defect in the skull remains. This procedure is preferred, since no decompression is indicated or desired. The scalp incision is readily closed in two layers. The drain is removed on the second and third days.

If the lesion is unilateral, the operation suffices. If bilateral, I prefer waiting two and one-half weeks, after which time a similar procedure is carried out on the opposite side. It is believed that better results will be obtained through employment of a small bone flap and the two-stage procedure in bilateral lesions. The bony defect is unnecessary and these small infants will not stand "too much operating." The results should be and are good in most instances.

SUBCORTICAL HEMATOMA

Subcortical hematoma may be traumatic or spontaneous. In those resulting from trauma, the blow may be severe and crushing, or fairly mild in character. The former, resulting from severe crushing blows, do not come within the scope of this paper. Only circumscribed lesions, whose bulk is entirely, or for the most part within the brain substance will be considered. The clot may be located in any part of the cerebral hemisphere and may destroy vital centers immediately, or they may remain comparatively quiescent and

unlocalized. They may occur singly or in patients with extradural or subdural clot, or both. About three years ago I operated upon a female patient who presented all three types of clot, one superimposed upon the other. The operation was carried out on the ninth day. The patient was in a stuporous condition accompanied by left hemiparesis. A rather long horizontal linear fracture extended through the midtemporal region of the right side, turned downward and bifurcated in the middle fossa. Therefore, the first burr hole was made directly over the fracture line. An ovoid extradural hematoma was removed from a low position over the sphenoidal lobe, rather deep in the middle fossa. Following its removal it was observed that the dura was blue, and when incised a typical, partly solid subdural clot was found and removed. It was then noticed that a small area of the surface of a subcortical clot presented itself on the surface of the temporal lobe. This subcortical clot was removed and proved to be solid and somewhat larger than a golf ball. It is probable that a laceration to the temporal lobe, with bleeding from the depth, occurred, and that both subcortical and subdural clots originated from this source. She made an uneventful recovery and at the present time is carrying on in her profession as a nurse.

For our purpose subcortical hematomas of spontaneous origin can be grouped in two classes, the one so well described by Craig, and the other seen in apoplexy, resulting from rupture of the lenticular artery. The pathology of the two lesions is so well understood that it will not be discussed.

Treatment. When the presence and localization of the clot in the first group is strongly suspected, a burr hole should be made in the overlying bone and a brain cannula of large caliber with multiple openings should be directed toward the position of the clot. When entered, a definite lack of resistance will be met. A major and frequently sufficient amount of the clot can be aspirated by suction, the cannula being used as a suction tip. In some instances the nature of the lesion may be determined while exploring for a brain tumor. If so, it should be evacuated without drainage.

Dr. Wingebach and I have aspirated a clot in several cases of apoplexy or stroke and without doubt saved the lives of patients who surely would have died. One appeared to be breathing his last gasp, and it was doubtful if he could be placed in an ambulance and taken alive to the hospital. When placed on the table he was in deep coma, with Cheynes-Stokes respiration. An enormous amount of clot, the color of blackberry jelly, was removed by suction and was followed by improvement in his condition. His first few weeks in the hospital

were rather stormy, and during this time, repeated tapping of the area was done. At each successive aspiration, a smaller amount of old blood was removed. His recovery was rather prolonged, but his life was saved. He now walks with spastic gait, but his mentality is clear.

From our experience with these cases we advise removal of a considerable portion of the clot in a selected group of cases of apoplexy, at first comprising otherwise hopeless patients. It is quite possible that the usefulness of this procedure may be extended and a considerable portion of the clot may be aspirated in other cases of apoplexy. Once the hemorrhage which occurs from the ruptured vessel has become arrested, the ends of the vessel become thrombosed. It is doubtful that recurrent bleeding would follow aspiration of a major portion of the clot. The syndrome is produced by sudden invasion of the brain substance by the rapidly forming clot, frequently of enormous proportions. The lessening of the bulk of these clots by aspiration might be as imperative as the removal of large extradural hematomas. The clot is always in the same position, varying only in size. A major portion of it can be aspirated through a burr hole made in the mid postparietal region.

To those surgeons particularly interested in the subject of intracranial hematomas, Munro's monograph on craniocerebral injuries, Vance's report of autopsies performed at Bellevue Hospital, and the splendid papers by Putnam and Cushing, Coleman, Craig, Dowman, Gardner, Grant, Gross, Kaplan, Kennedy and Wortis, McKenzie, Naffziger, and many other writers, which have appeared in the literature in the last decade, are recommended.

CONCLUSIONS

In this paper an attempt is made to present the subject of intracranial hematomas or blood clots and the allied conditions from a practical viewpoint, bearing in mind the general surgeons interrested in traumatic surgery, who in this time, because of their availability and the depletion of neurosurgeons will have to care for many of these patients. Very few, if any, original thoughts have been presented. No statistical or theoretical studies have been made.

Hematomas comprise one of the two major groups of intracranial lesions which can and should more often be relieved by timely and proper surgical intervention. Hemorrhage into the subarachnoid space and that associated with aneurysms have not been considered.

If this attempt may prove of any aid to the author and his colleagues in the restoration of these seriously injured patients to life and livelihood, it will have been justified.

THE SURGICAL TREATMENT OF PATIENTS WITH CRANIOCEREBRAL INJURY

JOHN RAAF, M.D.

Diplomate of the American Board of Neurological Surgery; Associate Clinical Professor of Surgery, University of Oregon Medical School

PORTLAND, OREGON

WHEN caring for a patient with craniocerebral injury a capital question which the physician must decide is whether surgical intervention is indicated. The factors so frequently creating a dilemma are first that the patient may have two or more co-existing intracranial conditions and second that one condition may be attended by signs which are supposed to be produced by another condition. Some surgeons[23] believe that operative intervention is indicated in not more than 5 to 10 per cent of patients with head injury. Pilcher and Angelucci[22] reported that among 373 patients fifty-three (14.2 per cent) were operated upon. Nineteen of the fifty-three patients died, an operative mortality of 35.8 per cent. They pointed out that among those who died following operation many had such extensive injuries that the operation played little if any part in the fatal outcome. It is Mock's[18] opinion that in a series of proved skull fractures if the physician operates upon less than 6 or 8 per cent he is overlooking operative indications and his mortality rate will be too high. If, on the other hand, the physician is operating upon more than 10 or 12 per cent of his consecutive run of patients with skull fracture, he is also in danger of increasing his mortality rate. In his series of twenty-nine operative patients eight died (27.5 per cent). Meredith[16] reported that among forty-two patients with craniocerebral injury who were operated upon ten died (23.8 per cent).

To propose an operation conscientiously upon a patient with head injury the surgeon must have in mind some purpose for which his operation is designed. The following are the most common surgical objectives in these cases: (1) Removal of intracranial hematoma (extradural, subdural, intracerebral); (2) removal of perforating foreign body; (3) repair of cerebrospinal fluid fistula; (4) exploration by trepanation; (5) reduction of increased intracranial pressure by decompression; (6) elevation of simple depressed fracture; (7)

débridement of compound wound, and (8) operation against complications (brain abscess, osteomyelitis of the skull).

The purpose of this paper is to discuss each of these indications for operation and to report results in a series of fifty-seven consecutive operative patients with craniocerebral injury who came under my care. Since the paper deals primarily with acute injuries, no patient who had been injured more than six weeks prior to operation was included in the.series.

REMOVAL OF INTRACRANIAL HEMATOMA

Removal of an extradural hematoma is usually a neurosurgical emergency. The classical signs of a free interval of consciousness, dilated pupil on the homolateral side, gradual development of hemiplegia and Jacksonian convulsions on the contralateral side are likely to be absent because damage to the underlying brain at the time of the accident may cloud the picture. Van Wagenen[25] and Gurdjian[10] both believe a lucid interval is the exception rather than the rule. It is particularly important to discover the presence of an extradural hematoma since lumbar puncture and dehydration therapy while indicated in cases of edema of the brain are contraindicated in extradural hemorrhage, for shrinkage of the brain in the presence of an extradural clot allows more space into which bleeding may occur. Whenever an extradural hemorrhage is suspected, one or more burr holes should be placed in the skull. It should be kept in mind that extradural hematoma is not always due to middle meningeal artery hemorrhage and therefore is not always found in the temporal region. In three of the four cases in this series the hematoma was encountered through a burr hole in the temporal region. In the fourth, trephining in the temporal region missed the hematoma and it was not until a burr hole was placed high in the parietal area approximately 5 cm. from the midline that the extradural clot was discovered. In two of the four cases the bleeding was from the middle meningeal artery, in one no single large bleeding point could be discovered and in the patient in whom the hematoma was high in the parietal area the source of bleeding was a vein running from the skull into the superior longitudinal sinus. Osteoplastic flaps were turned in three instances and in one the hematoma was removed through an opening rongeured in the temporal region. If the patient is in relatively good condition, I believe the turning of an osteoplastic flap is preferable to rougeuring away the bone because it allows more thorough evacuation of the clot, more adequate control of the bleed-

ing points, and less sacrifice of the cranial vault. In this series none of the four patients operated upon for extradural hematoma died.

The occurrence of subdural hemorrhage is more common than formerly thought. Kennedy and Wortis[15] reported than in their series there were seventy-two cases of acute subdural hematoma while there were only seventeen cases of extradural hemorrhage. They attempted to establish points of differentiation between the two types of hemorrhage. It was their opinion that subdural hemorrhage is likely to occur in all age groups while extradural hemorrhage is more likely to be found in the middle aged and young. In extradural hemorrhage the fracture line might overlie the site of the clot while in acute subdural hemorrhage the lesion more often is contralateral to the skull fracture or site of trauma. Extradural hemorrhage in their opinion is usually unilateral while acute subdural hemorrhage is more likely to be bilateral. In acute extradural hemorrhage, if there is paralysis, it is usually contralateral to the site of trauma while in acute subdural hemorrhage the side of paralysis is less predictable. The lucid interval is likely to be longer in acute subdural hemorrhage than in extradural hemorrhage. Kennedy and Wortis[15] reported that in a series of cases of acute subdural hemorrhage there was a mortality of 43.7 per cent after operation while the mortality in cases of extradural hemorrhage was 54.5 per cent.

Smith[23] advocated that operation for epidural hemorrhage be carried out as soon as the diagnosis is made and the period of shock is over, while he believed it is better to delay operation in cases of subdural hemorrhage for five to ten days if intracranial pressure can be controlled by conservative measures.

Among the fifty-seven patients in this series there were seven who were operated upon within six weeks of injury for subdural hematoma. In my experience many more patients with subdural hematoma come to operation when more than six weeks have elapsed from the date of injury than when less than six weeks have elapsed. Among the seven patients, bilateral subdural hematomas were found in one. Postoperatively, there were two deaths among the seven patients. One of the patients who died had a badly lacerated brain as well as an acute subdural hematoma; the other was in very poor condition at the time of operation. In six of the seven an osteoplastic flap was turned while only burr holes with washing out of the hematoma was employed in the treatment of one patient whose condition was desperately poor.

Gross intracerebral hemorrhages are not common in patients with head injury. Gurdjian[10] reported that in their series of about 400

operations for head injury there were only three instances of massive intracerebral hematoma. Courville and Blomquist[5] reported that among 439 fatal cases of head injury examined at necropsy thirty-six showed gross hemorrhages (in size not smaller than a cherry) within the brain. In my series there was one patient who not only had subdural and extradural hemorrhages but subcortical and intraventricular hemorrhage. Operation in this case was unsuccessful.

REMOVAL OF PERFORATING FOREIGN BODIES

Munro[19] has found perforating wounds of the skull either with or without injury to the meninges and brain are very rare in civilian practice. Any sharp instrument such as a knife or scissors blade, an ice pick or file may produce such a wound. Unless the instrument severs one of the large dural sinuses or a large artery so that severe hemorrhage occurs or penetrates deeply enough to injure vital structures at the base of the brain the outlook for recovery is good. There is, of course, always the possibility that infection will develop. The one patient in my series was a child who fell downstairs and drove the blade of a pair of scissors into her left frontal lobe. The blade broke off flush with the skull leaving a very small skin laceration. The parents, although they could not find the missing blade, had no idea it had penetrated the skull until a roentgenogram revealed its presence. Removal of the blade was followed by an uneventful convalescence.

REPAIR OF CEREBROSPINAL FLUID FISTULA

Meredith[16] believes that when there is an active cerebrospinal fluid leak from the nose, particularly if there is an intracranial aerocele, immediate operation is required as soon as the patient's condition will permit, the communication between the paranasal sinuses and the subarachnoid space being repaired with a fascial strip. Gurdjian[11] stated that when there is cerebrospinal fluid draining from the nose the possibility of intracranial infection is great; but that if the skull roentgenograms do not show fracture lines, particularly fragmentation at the base of the skull, conservative treatment can be undertaken. If conservative treatment is decided upon, the patient should be placed in semi-Fowler's position for four to six weeks; he should be as quiet as possible and never blow his nose. An antiseptic vasoconstrictor drug should be instilled in the nose three times daily. It was Gurdjian's[11] opinion that usually the drainage stopped within forty-eight hours. If the drainage persists for six to eight days or recurs after apparent cure, operative treat-

ment is indicated. I have had occasion to repair chronic but no acute cerebrospinal fluid fistulas. In my experience with six acute cases of cerebrospinal fluid rhinorrhea the drainage always stopped within a few hours after injury and under chemo-therapy infection from this source has not occurred.

EXPLORATION BY TREPANATION

There are patients who have sustained craniocerebral injury in whom the surgeon suspects that an intracranial hematoma may be present even though the classical signs which are supposed to be produced by these lesions are not present. If the patient has been given adequate conservative treatment and fails to show signs of improvement a burr hole exploration of the brain is indicated. In many of the patients the surgeon will find only cerebral contusion, laceration, edema and excessive subdural or subarachnoid fluid. However, an intracranial hematoma will be discovered sufficiently often to justify the frequent performance of this procedure. Some surgeons[11] advocate one trephine opening on each side of the head if subdural hemorrhage is suspected; if the hematoma is not found, encephalography rather than more openings in the skull is indicated. Burr holes can be so easily and quickly made in the skull I generally place three or four on each side. The patient's head is supported in the cerebellar region so that sterile draping of both sides of the head can be obtained. If a hematoma is found on one side, that side is turned up and the remainder of the operation carried out.

In the series being reported twenty-nine patients had burr hole exploration. Included among these twenty-nine patients are those also classified under "Removal of intracranial hematoma" and "Reduction of increased intracranial pressure by decompression" because operations on patients in both these latter groups were started as burr hole explorations and then converted into some other kind of operation the type depending upon what the burr hole exploration revealed. In twelve of the twenty-nine patients intracranial hematoma was found and the operation then directed toward its removal. In six patients burr hole exploration was followed by an operation intended to relieve increased intracranial pressure and in the remaining eleven patients the operation terminated with burr hole exploration only. Among these eleven patients two were found to have a large amount of subdural fluid. Seven had excessive subarachnoid fluid and in two there was no increase in either the subarachnoid or subdural fluid. Of the eleven patients who had burr

hole exploration of the brain which was not followed by any other type of operation four died and seven recovered. The mortality rate in this group is high because I have made it a rule never to let a patient expire without trephining the skull unless he has a head injury of such severity that his case is obviously hopeless. The occasional discovery of an intracranial hematoma in a patient who is practically moribund and who subsequently recovers after operation has made this effort worth while. Two of the patients who had a marked increase of subarachnoid fluid showed very rapid improvement following its release. It is my opinion that the release of subarachnoid as well as subdural fluid in some of these cases justifies the operation even though no intracranial hematoma is found. It has been noted that in some of these cases there was marked edema of the scalp following trephining, indicating that an excessive amount of fluid was draining through the burr holes.

REDUCTION OF INCREASED INTRACRANIAL PRESSURE BY DECOMPRESSION

The majority of surgeons handling patients with head injury now believe that operations devised primarily for decompression of the brain are not advisable. While the decompression will increase the size of the intracranial cavity, frequently the swollen brain herniates through the opening causing tears in the brain and hemorrhages. In six of the twenty-nine patients who had burr hole exploration this procedure was followed by an attempt to reduce the intracranial pressure through decompressive measures. In all these cases it had become obvious that conservative methods were destined to failure; and even though it was known that "the dice of the gods were loaded," decompression was performed. Two of the patients had unilateral subtemporal decompression, accomplished by enlarging a burr hole with the rongeur; three had bilateral subtemporal decompression done in the same manner. One had a large temporoparietal block of bone removed by means of the burr and Gigli saw. Operation in none of these six cases saved the patient's life. This bears out the opinion that decompressive operations of this sort are of little or no value.

Briesen and Jones[2] suggested that increased intracranial pressure following head injury can be relieved by placing a drain in the cisterna magna and they reported one successful case. Ody[20] reported a similar operation. It has been noted on numerous occasions that a patient who drains cerebrospinal fluid from his ear or nose, if he does

not develop infection, recovers more rapidly than the one who does not have a loss of cerebrospinal fluid. This might indicate that the suggested operations of Briesen and Jones[2] and Ody[20] are of value.

ELEVATION OF SIMPLE DEPRESSED SKULL FRACTURES

Patients with simple depressed skull fractures should be operated upon for cosmetic purposes and to relieve pressure upon the brain. In most instances the elevation of the depressed fragments is not a surgical emergency although it should be carried out before the fragments become too firmly fixed in malposition. Walker[27] stated that the indications for early operation are continued convulsions which could not be relieved by dehydration and decline in the patient's condition in the absence of some other cause. Glaser and Shafer[8] concluded that early elevation of the depression did not prevent late sequelas. It seems that epilepsy is more likely to be due to the formation of a cerebral cicatrix than the irritating effects of a depressed bone fragment. Among six patients in this series operated upon for the elevation of simple depressed fracture there were no deaths.

DÉBRIDEMENT OF COMPOUND WOUNDS

A high proportion of patients suffering head trauma will sustain compound wounds. This is particularly true among war casualties. Although the maxim "treat shock first" is continually ringing in one's ears, only a small percentage of patients with craniocerebral injuries are in shock on arrival at the hospital. Meredith[16] stated that less than 10 per cent of any large series of patients with head injury of all types are in shock.

Certainly as soon as the patient's condition will permit, a débridement of the wound should be performed provided adequate facilities are at hand. Grant[9] has pointed out that two lessons seem to stand out from the first World War: (1) Patients with cranial trauma can be transported with greater safety before operation than immediately afterward; (2) when the patients with craniocerebral injury are operated upon by specially trained men in properly equipped centers they do better than when they are assigned to the general surgeon. Jentzer[14] remarked upon the importance of having complete neurosurgical equipment and thought that the necessary quiet for a neurosurgical operation could not be found closer than twenty-five kilometers behind the battle line. He thought that the patients following operation should not be transported for at least fourteen

days. Tönnis,[24] in speaking of the treatment of patients with brain
injury sustained during the campaign against Poland, stated that
operations attempted in unsuitable conditions made the patients
worse. Those who had the good fortune to be transported to a base
hospital were the only ones who did well, and those who were trans-
ported by air did the best. Cairns[4] agreed that if the patient's condi-
tion was unfavorable or adequate operating facilities were not at
hand it is better to delay operation. He stated that brain tissue
resists and localizes infection as well if not better than other tissues.

Horrax[12] suggested that three prophylactic measures should be
instituted in the treatment of patients with war wounds of the brain:
(1) Soldiers should go into action with closely cropped hair; (2)
tetanus toxoid should be given to all soldiers and civilians likely to be
involved in enemy actions; (3) the sulfonamides should be given by
mouth as soon as possible after any kind of compound injury has
been received.

When a patient with a compound skull fracture is first seen, the
hair about the wound should be clipped or shaved, one of the sulfon-
amide drugs sprinkled in the wound and bleeding from the wound
controlled by packing with gauze. These procedures are particularly
important if for any reason some hours are likely to elapse before a
careful débridement can be done.

The best anesthetic for the débridement is novocain although if
the patient is exceptionally violent avertin or pentathol sodium may
be necessary. The patient can be spared some pain if the surgeon will
shave and clean the scalp at a considerable distance from the wound.
Novocain can then be infiltrated into the scalp around the wound at
some distance from the edges. Following this, cleansing of the wound
edges with tincture of green soap, ether, alcohol, and tincture of
merthiolate will be painless. When the region is prepared, one should
drape a large area because it may be necessary to extend the skin
incision for some distance to expose adequately the defect in the
bone. Ragged skin edges are excised and the knife discarded. Hair
and débris are removed from the wound and copious quantities of
warm saline solution employed for washing the wound. The extension
of the skin incision, in order to secure adequate exposure, may be
done in several ways. If there is loss of scalp, the tripod incision or
the three-legged (Isle of Mann) incision may be employed to facili-
tate co-aptation of the wound edges. Where the scalp wound is
small, a horseshoe-shaped skin flap with the wound of entry in the
middle may be turned as suggested by Winslow.[28]

In order to elevate the depressed bone fragment it may be neces-
sary to place a burr hole adjacent to the edge of fracture and cut
along the fracture line with the deVilbiss. The depressed bone is
lifted out in as large pieces as possible; these are taken by the surgical
nurse and thoroughly cleansed with saline solution. When the
depressed bone fragment is lifted out there may be sharp bleeding if
one of the venous sinuses has been torn by the bone edge. This bleed-
ing can be controlled with a muscle stamp. The removal of all cracks
in the bone in which infection may track has been suggested.[25] It
seems to me that this would be impractical unless there is gross
débris in the fracture line since in many patients linear fractures
radiate out for several inches from the area of depression. While
most surgeons nibble away the bone edges to remove the depressed
fragment and gain adequate exposure Burckhardt,[3] Garcin and
Guillaume,[7] and Vincent and Lafon[26] turned osteoplastic flaps to
expose the penetrating wounds. Ducuing[6] found that in most cases
fractures in the skull were so extensive that the flap broke to pieces
when this method was employed.

If the dura is intact and there is no evidence of hemorrhage under-
neath, as indicated by a tight dura or discoloration, it should not be
opened. When the dura is lacerated and the brain substance
damaged, the necrotic brain tissue is removed with the suction
apparatus, care being taken not to encroach any more than necessary
upon nerve pathways which are still intact. Also one should be careful
not to open into a ventricle or basal cistern since this increases the
danger of infection. Bullet and bone fragments are removed under
direct vision if they are accessible. A rubber catheter is excellent for
exploring the track in the brain. By means of the sucker the track is
converted into a smooth-walled cavity. If the bullet has traversed the
brain to an inaccessible position, an attempt should not be made to
remove it. Ascroft[1] analyzed 317 cases of gunshot wounds of the head
(patients with wounds of the cerebellum were excluded from this
series) sustained during the 1914 to 1918 war. He found that 34 per
cent developed epilepsy but that there was no evidence that removal
of metallic foreign bodies from the brain diminished the liability of
this complication. He thought prolonged wound sepsis favored the
subsequent development of epilepsy both in those cases in which the
dura had been penetrated and in those in which the dura had
remained intact.

The dura should be closed if possible. When there is a communi-
cation between the subarachnoid space and one of the paranasal

sinuses, it is imperative that a tight closure be affected, taking if necessary fascia lata, fascia from the temporal muscle or periosteum from under a scalp flap to affect the plastic closure. If the compound fracture is over the vault and the dura cannot be adequately closed, a small opening in the dura is not of much importance if a good skin closure can be affected. In these cases the opening in the dura can be covered by a nonadhesive membrane manufactured for such a purpose.

In civilian practice the pieces of the skull which have been thoroughly washed with saline solution can be replaced and a firm closure obtained. It may be necessary to hold the fragments together with sutures passed through drill holes in the pieces of bone. The skin is closed with interrupted No. oo plain catgut to the galea and interrupted fine silkworm sutures to the skin. If the laceration is in the forehead, a fine Kal-dermic (6-o) or horsehair suture is used in the skin. No drains are left in place unless there is evidence of infection at the time the débridement is performed.

The local use of the sulfonamides in the wound has come in for much discussion in the last few years. Mitchell, Logie and Handley[17] in commenting on experiences in the British campaign in Libya stated that the use of these drugs cannot take the place of careful treatment of war wounds. It is still a debatable question which of the drugs is most effective locally in the brain and which is least likely to produce an untoward effect. Experimental work[21] which may help to solve this problem is now in progress. Hurteau[13] found that after local application to cerebral wounds the several drugs of the sulfonamide group show a difference in the rate of absorption. According to him sulfanilamide is the most rapidly absorbed, sulfathiazole the second, sulfadiazine third, and sulfapyridine the least rapidly. He suggested the mixture of several of the drugs, for example sulfanilamide and sulfadiazine. A high concentration of sulfanilamide would occur immediately while a high concentration of sulfadiazine would persist for a time to cope with latent infection.

In my series of nineteen patients who sustained compound skull fracture thirteen had laceration of the dura and in seven of the nineteen there was loss of brain substance. The time which elapsed from injury to operation ranged from three to thirty-four hours, the average elapsed time being ten and one-half hours. While one likes to operate upon these patients within six hours of the time of injury, many patients in this series were sent in from some distance so the average time from injury to operation was well beyond six hours.

Thorough débridement was carried out in each case and in fourteen patients the pieces of bone were replaced so no defect would remain in the skull after healing. In fifteen the skin edges were closed tightly while in three a small Penrose drain and in one a small wick of gauze was placed underneath the skin. One patient with an extensive laceration of the brain died seventy-two hours after surgery and the other eighteen recovered. All wounds in the eighteen patients who recovered healed without infection and there was no evidence of infection in the wound of the patient who died. In two instances there was a slight amount of cerebrospinal fluid drainage for a few days. Sulfanilamide or sulfathiazole was sprinkled in the wounds of seven patients but it is difficult to ascertain the efficacy of the sulfonamide drugs applied locally in these cases since infection did not occur in any of the patients.

OPERATIONS DIRECTED AGAINST COMPLICATIONS

A complication such as brain abscess, osteomyelitis of the skull, or meningitis may follow craniocerebral injury. Usually the osteomyelitis is not the rapidly progressive fulminating type which frequently occurs secondary to infection in the paranasal sinuses. It cannot be overemphasized that the best treatment for osteomyelitis of the skull and brain abscess is preventive. No craniocerebral injury or even simple scalp laceration should be neglected. All should be thoroughly cleansed, repaired and bandaged.

In this series one patient who sustained a fracture through the base of the skull with the subsequent development of osteomyelitis of the cribriform plate and abscess underneath the frontal lobes was operated upon but did not survive. Another patient had a scalp laceration sutured by his local doctor who did not realize a compound skull fracture was present. Subsequently a subdural abscess formed. Following removal of the bone fragments, drainage of the abscess and vigorous chemotherapy he finally recovered.

CONCLUSIONS

1. Twelve patients with intracranial hematoma were operated upon and nine of the twelve recovered (mortality 25 per cent).

2. Patients who do not improve after adequate conservative treatment should have burr hole exploration of the brain for there is no other way to exclude conclusively the possibility of an intracranial hematoma.

3. Removal of portions of the skull for the primary purpose of reducing intracranial pressure did not prove effective.

4. Careful débridement of compound craniocerebral wounds will be attended by a high percentage of recoveries. In this series eighteen out of nineteen patients with compound wounds recovered (mortality 5.3 per cent).

5. The mortality rate for fifty-seven patients with craniocerebral injury operated upon within six weeks of injury was 26.3 per cent.

TABLE I

OPERATIONS UPON FIFTY-SEVEN PATIENTS WITH ACUTE CRANIOCEREBRAL INJURY *

Purpose of Operation	No. of Patients	Deaths
Removal of intracranial hematoma		
Extradural	4	0
Subdural	7	2
Intracerebral	1	1
Removal of perforating foreign body	1	0
Exploration (burr holes)	11	4
Reduction of increased intracranial pressure (decompression)	6	6
Elevation of simple depressed fracture	6	0
Débridement of compound wound	19	1
Against complications		
Brain abscess	1	1
Subdural abscess	1	0
Total	57	15 (26.3 per cent)

* All of the patients in this series were operated upon within six weeks of the time of injury.

REFERENCES

1. ASCROFT, P. B. Traumatic epilepsy after gunshot wounds of the head. Brit. M. J. 1: 739–744, 1941.
2. BRIESEN, HANS V. and JONES, EVAN M. New operations for relief of increased intracranial pressure in head injuries Am. J. Surg., 50: 126, 1940.
3. BURCKHARDT, H. Friedrich method of wound excision and customary partial wound excision particularly in cranial and cerebral injuries. Deutsche Ztschr. f. Chir., 254: 249, 1940.
4. CAIRNS, H. Gunshot wounds of the head in 1940. J. Roy. Army Med. Corps, 76: 12, 1941.
5. COURVILLE, C. B., and BLOMQUIST, O. A. Traumatic intracerebral hemorrhage with particular reference to its pathogenesis and its relation to "delayed traumatic apoplexy." Arch. Surg., 41: 1, 1940.
6. DUCUING, J., D'HARCOURT, J., GRINO, A. and FOLCH, A. General principles in the treatment of craniocerebral wounds in war. Rev. de Chir., 77: 625, 1939.
7. GARCIN, R. and GUILLAUME, J. Notes on the treatment of head wounds in war and the results obtained by the use of modern neurosurgical methods in advanced zone hospitals. Mém. Acad. de chir., 66: 124, 1940.

8. GLASER, M. A. and SHAFER, F. P. Depressed skull fractures; value of surgery; sequelae; an eight year follow-up study of ninety-one patients. *J. A. M. A.*, 113: 2111, 1939.

9. GRANT, FRANCIS C. Treatment of cranial injuries in war. *Surg. Clin. North America*, 21: 1623, 1941.

10. GURDJIAN, E. S. Traumatic intracranial hemorrhage. *Indust. Med.* 10: 412, 1941.

11. GURDJIAN, E. S. Pathology and surgical management of acute head injury. *South. Surg.*, 10: 711, 1941.

12. HORRAX, GILBERT. The treatment of war wounds of the brain. *New England J. Med.*, 225: 855, 1941.

13. HURTEAU, EVERETT F. The intracranial use of sulfadiazines: experimental study of the histology and rate of absorption. *Canad. M. A. J.*, 46: 15, 1942.

14. JENTZER, A. Traumatismes craniens et plaies cranio-cérébrales par projectiles. (Head injuries and cranio-cerebral wounds caused by projectiles.) *Schweiz. med. Wchnschr.*, 70: 648, 1940.

15. KENNEDY, F. and WORTIS, H. Epidural hemorrhage; study of 72 cases of hematoma and 17 cases of hemorrhage. *Surg., Gynec. & Obst.*, 63: 732, 1936.

16. MEREDITH, J. M. The management of head injuries *West Virginia M. J.*, 34: 289, 1940.

17. MITCHELL, G. A. G., LOGIE, N. J. and HANDLEY, R. S. Casualties from the western desert and Libya. *Lancet*, 1: 713, 1941.

18. MOCK, HARRY E. Management of skull fractures and cerebral injuries. *J. Iowa M. Soc.*, 27: 525, 1938.

19. MUNRO, DONALD. Early diagnosis of craniocerebral injuries. *Am. J. Surg.*, 56: 3, 1942.

20. ODY, F. Atlanto-occipital evacuative trepanation in contusion of the brain. *Arch. Neurol. & Psychiat.*, 28: 112, 1932.

21. PILCHER, COBB. The principles governing the diagnosis and treatment of acute cranio-cerebral injuries. *South. Surg.*, 10: 783, 1941.

22. PILCHER, COBB and ANGELUCCI, RALPH. Analysis of three hundred and seventy-three cases of acute craniocerebral injury. *War Medicine*, 2: 114, 1942.

23. SMITH, GERALD W. The diagnosis and treatment of head injuries. *U. S. Naval Med. Bull.*, 38: 447, 1940.

24. TÖNNIS, W. Die Behandlung der Hirnverletzungen aug Grund der Erfahrungen im Feldzug gegen Polen. (The treatment of brain injuries based on experiences in the campaign against Poland.) *Deutsche med. Wchnschr.*, 66: 57, 1940.

25. VAN WAGENEN, WILLIAM P. The rational of certain procedures in the treatment of head injuries. *Neurosurgical Ward Rounds*, 2: 12, 1940.

26. VINCENT, C. and LAFON, J. L. Neurosurgical treatment of craniocerebral injuries. *J. de méd. et chir. prat.*, 3: 87, 1940.

27. WALKER, DUDLEY P. Fractures of the skull and associated brain injuries. *Pennsylvania M. J.*, 45: 472, 1942.

28. WINSLOW, PHILIP. Penetrating wounds and foreign bodies of the brain. *Neurosurgical Ward Rounds*, 2: 1, 1941.

DISCUSSIONS OF PAPERS OF DR. KING AND DR. RAAF

DONALD MUNRO (Boston, Mass.): I have been privileged to receive copies of Dr. King's and Dr. Raaf's papers, and I am sorry that time did not permit the authors to read them in detail, because you would have been well repaid by having heard them read in that way. I recommend that all of you read them in detail when they are published.

The contents of these papers do not allow for any discussion. Everything that is said is without doubt true, and I am in complete agreement with the subject matter in both papers, except for certain minor details.

Because of the impossibility of discussing an undiscussable paper, it seemed to me that I might avail myself of this opportunity to present to you certain unpublished data in regard to some of the high spots of these problems of intracranial hematomas and compound fractures of the skull.

I should like to speak first about extradural hematomas. I do not look upon extradural hematomas with the same equanimity as Dr. King and Dr. Raaf do. In my hands, I am of the opinion that extradural hematomas presented one of the most difficult problems of diagnosis that the traumatic neurosurgeon has to face. The classical syndrome by which the diagnosis is said to be possible has, in my hands, been so rare as to be cause for calling the house officers and other members of the staff around, so they could see an example of this curious, interesting phenomenon.

In a group of thirty-seven extradural hematomas, in ten cases the clot resulted from venous hemorrhage. I should like to emphasize that, because I think that is not very well recognized, and I think that it is important because it is in these cases, in particular, that the patients live a relatively long time between the infliction of the wound and their death.

In regard to the diagnosis, in my group of cases only 40 per cent showed a lucid interval. That is considerably less than one would be led to expect from reading the ordinary surgical textbooks; and I commend to you the fact that you will probably see few extradural hematomas that show the pathognomonic triad with the lucid interval between two periods of unconsciousness, and you will see patients with extradural hematomas who remain constantly unconscious from the time they received the blow until you operate upon them.

In support of that, 50 per cent of my patients were unconscious on admission, and 92 per cent had bloody cerebrospinal fluid, which led us to conclude that all of those patients had at least a contusion of the brain with the associated uncounsciousness that goes with contusion of the brain.

In regard to subdural hematomas, these are a very common complication of craniocerebral injuries. They are, in the acute cases, in my experience, diagnosable only by exploratory trephination, and in connection with that, I think that it is quite possible that as we get more experience and as you general surgeons begin to take up part of this load, you will find, and we will find, that multiple exploratory burr holes will be more and more common. By multiple exploratory burr holes I mean not two burr holes, one on either side, but I mean many burr holes done in all areas of the skull until the hematoma is actually demonstrated or until an adequate spotty exploration of the contents of the skull has been carried out.

In connection with that, it has recently been recommended that this exploration be carried out with the patient in the cerebellar position so that

burr holes can be placed over the posterior fossa as well as over the two lateral fossae, and in that way the cerebellar subdural or extradural hematoma be identified.

In my experience, I have had 310 patients with subdural hematomas. Practically all of them have been operated upon, and in all of them the subdural hematoma has been the major complicating factor. One hundred ninety-four of these 310 cases were associated with major brain injuries, and I believe that this type of subdural hematoma, the subdural hematoma that is caused by the associated brain injury, is far and away the most important variety of subdural hematoma and is the one that yields most readily to the immediate operation done within a short time after the entrance of the patient into the hospital. In this group my mortality has been 40 per cent. These mixed hematomas, if they are not operated upon, go on to an end result of a cystic collection of fluid which is free from the subdural space. That has been described formerly as a hydroma. I prefer to consider it a hematoma, because they all start out as a mixture of blood and cerebro-spinal fluid, and because a hydroma requires by definition that the fluid be contained within a sac. Hydroma as a name could be more properly applied to the end result of the solid subdural hematomas.

There were seventy-one of these. Of the solid clots, there were only forty-five. These are the clots that were described by Cushing and Putnam.

Just a word about compound fractures. The greatest problem, probably, in compound fractures today is when to operate upon the compound fracture and what kind of first dressing to apply..The latter is particularly true because of the present use of the sulfonamide products. In an endeavor to find out when to operate upon compound fracture wounds, I have recently gotten together statistics from fifty-six cultures made on fifty-six compound fracture wounds. It was interesting to note that contamination, although doubtless present in every wound, was culturable in only 55 per cent during the first twenty-four hours, and in only 66 per cent during the first forty-eight hours, whereas after forty-eight hours, 91 per cent of these wounds showed easily culturable bacterial contamination. It seems to me in view of this, and in view of my experience, there is never any hurry about operating upon compound fractures of the skull, provided you do the operation within the first forty-eight hours of the receipt of the wound, and provided, and this is equally important, you do not manipulate the wound at the time of the first dressing.

In connection with that, I would like to say that the results published by Cloward, in Pearl Harbor, it seems to me, are dangerous, because he suggests that at the dressing station the patients with compound fracture wounds not only had their hair clipped but that the scalp was shaved and the wound was scrubbed at that time. This I consider to be a very dangerous procedure because I am sure under that amount of manipulation there will be a wider spread of contamination into the wound than would otherwise exist if the wound were left alone until the major débridement is done.

In regard to the use of sulfonamide products in these wounds, there is no question but that is a very valuable adjunct to our surgical procedure in dealing with compound fractures. However, do not use sulfathiazole on the surface of the brain. Recently a series of cases have been reported—and there are some others that have not been reported—in which the use of sulfathiazole on the surface of the brain, both experimentally and in the human being, has produced epileptiform seizures which were traceable directly to the use of that particular drug. If you want to use the sulfonamides in the wounds themselves, use sulfanilamide or sulfadiazine powder.

Of course, the sulfonamides should be given by mouth from the first time that you see the patient.

In closing, I would like to congratulate this society and congratulate the two readers on having brought this important general surgical problem to the notice of general surgeons, and taken it away from the narrow specialty, where it has been resting for the most part, to the present. These problems of craniocerebral surgery are general surgical problems. More and more of the general surgeons are going to have to face them and deal with them, and it is well that that should be brought to the attention of a general surgical society such as this.

WILLIAM JASON MIXTER (Boston, Mass.): Dr. Munro said he was not going to discuss these papers. I think he did a pretty good job of it, and I do not think he left me much to discuss.

They are good papers, and they are papers of great interest to a group of men who are handling the surgery of trauma, not only for the reasons that Dr. Munro has given, but also due to the fact that even if you do not want to handle these cases yourself, sometimes you are forced to, just the same as Dr. Munro or Dr. King or I do when we go to some little hospital out in the sticks around Massachusetts to see a patient with a severe head injury, and there we find a tibia scratching around in the sheets and nobody has done anything to take care of it before we got there. That happened to me within the last two years, and the compound fracture of the tibia was a more important feature than the head injury.

The war brings up the same problem. Even if we wanted to put a neuro-surgeon in every front-line hospital, it could not be done. There are not enough of them. The result is that the general surgeons must pick up a considerable group of these cases and handle them, and handle them intelligently. For that reason, I commend these papers to you, as Dr. Munro already has.

JOHN S. HODGSON (Boston, Mass.): I have found Dr. King's and Dr. Raaf's papers of great practical interest. Traumatic hematomas, whether they are extradural or subdural, or the rare subcortical ones, plus compound and depressed skull fractures and complicating spinal fluid leakage from cranial wounds and orifices, intracranial aerocele and infection of the scalp, bone, meningeal spaces and brain itself, represent the conditions calling for

cranial or intracranial surgery. It seems to me that the general surgeon can be of definite help in traumatic neurosurgery in these times.

The papers have been so well given and completely discussed that it is difficult to know just how much to say. I am going to limit my remarks except to say that the traumatic extradural hemorrhages are rather rare, comprising a very small percentage of the cases of head injury, but they are so serious that their rarity makes them all the more important. Whether or not one uses a burr hole and enlarges it, or uses a bone flap, is somewhat unimportant, probably. I think that most of us probably simply remove enough bone to get at the bleeding point, which is usually under the temporal muscle, but if there is some question as to the location of it, it may be better to make a bone flap.

While he did not speak of it, Dr. King wrote in his paper about sometimes needing to tie off the external carotid artery in cases in which the extradural hemorrhage was not easy to locate, or in cases in which it was extreme in degree and the patient's condition might be such that time was very valuable. I think in such a case it might be all right to tie off the external carotid artery, but that must be a rather rare necessity, of course.

I think anyone doing the surgery of extradural hemorrhage must be prepared to follow through to the point where the hemorrhage is coming from, even if that involves going to the base of the brain, as far as the foramen spinosum, and plugging that foramen with cotton. I think it is better to do that than to do the ligation of the external carotid, although the latter may be necessary in rare instances.

In regard to the subdural hematomas, those, of course, are much more common and so much has been said about those that I am not going to attempt to add anything at this time.

The subcortical hematomas are extremely rare, and yet they do occur, and when one has decided to operate and does operate, and does not find what he thinks he is going to find, namely, a subdural hematoma, he should be prepared to look at least locally for a possible subcortical hematoma. That was brought home forcibly to me again recently when I operated upon an elderly person whom I suspected of having a subdural hematoma, which he did not have. He had no localized signs at all but the cortex appeared a little abnormal in that region, and on tapping it, I found a rather deep and large subcortical hematoma. Therefore, one should be eager enough to follow through, to look for a possible subcortical hematoma. It will be rare, but it occurs just often enough to necessitate one's always keeping it in mind and looking for it every now and then. Of course, it may occur along with extradural or subdural hematoma as well.

Regarding the repair of dural rents in cases in which there may be spinal fluid leakage from a head orifice, I should say that that is a pretty major operation, and it would seem as though the general surgeon would do well not to take on a case of that kind, deliberately, at least, because it does

involve pretty complete experience with neurosurgery. It means making a large scalp incision, turning back a bone flap, looking deep in the cranial fossa, finding the rent in the dura and then sewing it up. It is not easy to sew up rents in the deep dura. You are told that all you need to do is sew the edges together, but they are so thin and friable that they cannot be sewn together easily, so that you either have to use a piece of fascia, which often does not work well, or a piece of muscle. I have found that a piece of muscle is rather good to put in between the edges of the dura that will not come quite together. If one will stitch the edges together with a piece of muscle in between, he will get a good closure which he otherwise could not get at all.

Also, it is a good plan sometimes to take a piece of muscle and cover over places like the cribriform or some other place where there is a fracture in the base of the skull. That will do what nothing else can do.

I had an interesting case recently which I think it will be worth while mentioning. It was a child of eleven who came into the Massachusetts General Hospital with the history of having had a head injury at the age of four, seven years previously. There had been bleeding from the ear which had stopped, and the child had apparently been all right, as far as we could find out from the family, during the succeeding seven years. Just before coming in, she developed a spontaneous leakage of spinal fluid from the ear on the side that had leaked seven years before. She had come into the ear and nose clinic and was transferred to the neurosurgical service. X-ray showed a fracture in a portion of the temporal bone. We operated, took out a large piece of bone in the temporal region, made drill holes and lifted out the dura and found the rent in the dura. We could see the fracture line in the bone still. The dura had funneled into the fracture line in the base of the skull. The dura was pushed back, the fracture line in the skull was sealed off with bone wax, and the dura edges were brought together with a little difficulty because they were thin and friable and I had to put a piece of muscle in between and close it in that fashion. Healing was complete and she got along very well.

That to me was a rather unusual case. I have never personally happened to have to repair a cerebrospinal fluid orrhorrhea before. They all leak, if they leak at all, for a few days and then stop. The repair was done in the same fashion that you would repair a cerebrospinal rhinorrhea.

One other case that interested me a good deal in regard to a repair was that of an elderly man who had had a severe head injury. He had been kicked by a horse. It had made a straight up and down scalp wound. He had been seen by a local physician who took charge of him and did the original surgery. At the time of the injury, this horse that kicked him kicked out a large piece of skull in the left temporoparietal region. At the time the man was picked up, the piece of bone was seen in the street and picked up along with the man and taken to the hospital. The scalp wound was sutured;

the dura was not lacerated much and he had not suffered much intracranial damage. The doctor simply sewed up the scalp wound and kept the piece of bone in saline.

When I saw the man a few weeks after, the wound was not quite healed, and we waited three weeks more. He was transferred to the Massachusetts General Hospital and the piece of bone came along with him. That was sterilized, the wound was reopened and the piece of bone put in. That operation was finally done about eight weeks after injury. It did not completely fill in the space because a little had been broken out in small pieces in the low temporal region, but that was put back in and sutured in with small drill holes and suture material, and the wound healed well and has remained healed since that time, a matter now of several months.

JOSEPH E. J. KING (closing): I necessarily had to read my paper in such a sketchy manner that I did not cover all the points, and I believe if Dr. Raaf will wait until he reads my paper, he will find he agrees with me, and I certainly agree with him except about the bone flap. You must remember that these people are sick and you cannot fool around and turn up nice bone flaps. What you want to do is to get the blood clot out and get the patient back in bed, and I can do it through one or two holes much quicker than by making a bone flap. If you make a bone flap you have to make a lot of holes, too.

Most blood clots come from the middle meningeal injuries.

It is true that ligation of the external carotid will very seldom be used, but you should not send a patient back to bed when you cannot see where the blood is coming from. You know how it comes out like a waterfall sometimes. Pack it and make sure the packing is secure, but rather than risk it, I would ligate the carotid. I had to do that for a patient with unilateral nose bleed. He would have died otherwise. He had had five transfusions and we could not stop the hemorrhages, and I finally had to ligate.

Dr. Munro said something about Dr. Raaf and myself viewing these cases with equanimity. No, we do not do that. I know they are serious cases and I look at them very seriously.

In the case of venous hemorrhage, of course, especially in the posterior fossa, I agree with Dr. Munro that you must have exploratory trephination all over, not as many openings as in a chair back, but several openings are much better than to send a fellow back and not find it. I even put in the paper what a humiliating feeling it is to operate upon some patient for a clot which you do not find, and when he comes to autopsy examination there is a big one you missed.

JOHN E. RAAF (closing): On this matter of the bone flap, I will agree with Dr. King that if you stand by and watch the patient with an extradural hematoma until he is in a serious condition, the best method of treatment is to put in a burr hole, enlarge the burr hole and suck out the clot. However, if the patient is in good condition, I would rather turn a bone flap,

because even though a single bleeding point may start the hemorrhage, as the hematoma develops and strips the dura away from the bone, numerous bleeding points will start. There are blood vessels running between the dura and the bone which will start to bleed as the clot develops, and you can more adequately control these if you have a large flap.

I will agree with Dr. King that I did not mean to create in Dr. Munro's mind the impression that I thought the diagnosis in extradural hemorrhage is easy. It is difficult, and I think that is the reason many of us are coming to the point where we put multiple burr holes in the skull. It has been advocated lately by at least one surgeon that one burr hole should be placed on each side and if the hemorrhage is not found, one should resort to other studies, such as encephalography. I disagree. I think multiple burr holes are indicated, and sometimes I operate upon patients in a sitting position so that numerous burr holes can be placed in the skull, including the occipital area.

TRAUMATIC RUPTURE OF THE SPLEEN*

LOUIS C. ROETTIG, M.D.,
Resident in Research Surgery,
Ohio State University

W. D. NUSBAUM, M.D.
Attending Surgeon, Lancaster
City Hospital, Lancaster

AND

GEORGE M. CURTIS, M.D.
Chief of Research Surgical Service,
Ohio State University Hospital

COLUMBUS, OHIO

TRAUMATIC rupture of the normal spleen is not the clinical rarity it was formerly thought to be. Its increased incidence is both real and apparent. On the one hand, this is due to better diagnosis; on the other it is accounted for by the year-to-year increase in automobile and industrial accidents. It is also important to note that in reports thus far available from England ruptured spleen is high on the list of abdominal injuries incident to blast.

The exact incidence of traumatic rupture of the spleen is difficult to determine. That it is higher than is commonly recognized is one of our fundamental conclusions. That the diagnosis is too frequently missed we know from experience at our own institution. This is one of the prime reasons for again calling the attention of the profession to this result of trauma. Working as we do in a general hospital which serves private patients as well as a teaching institution for the medical school, we have the opportunity to observe the methods of a considerable group of surgeons in the management of accident cases. During the present year, two cases (Nos. 19 and 20) of traumatic rupture of the spleen remained undiagnosed until necropsy.

In reviewing the pertinent literature one is impressed by the large number of papers reporting one or two cases of splenic rupture, indicating that many surgeons still consider it rare enough to warrant a paper composed of a single case report. That the true incidence is far greater is indicated by the report of Wright and Prigot.[48] They found traumatic rupture of the spleen in one out of every 666 accident cases of all kinds admitted to the Harlem Hospital in New York City.

At the University Hospital our incidence has been computed to be one out of every 920 accident admissions of all types.

This report is based on a series of twenty-two cases of traumatic rupture of the spleen occurring in the city of Columbus, Ohio, during the years 1929 to 1941 inclusive. This period has been chosen because it begins with the adoption of uniform record library methods throughout the city. The cases reported are all those recorded in the files of the city's six hospitals during the period. Our own cases total eight in number and represent one-third of the group. We believe that this group represents an accurate picture of the occurrence, recognition and management in a fairly typical mid-western city of 300,000 inhabitants. It affords a good basis from which to appraise both the mistakes and successes in the present day surgical management of traumatic rupture of the spleen.

ANATOMY AND PHYSIOLOGY

A brief review of the anatomy and physiology of the spleen is of considerable help in a better understanding of the pathology and treatment. The spleen is a vascular, lymphoid organ measuring normally about 12 by 7 by 4 cm. and weighing about 200 Gm. Its tissue is soft and friable. Lying as it does between the fundus of the stomach and the diaphragm the spleen is normally protected by the lower portion of the thoracic cage. It is almost entirely surrounded by peritoneum and is held suspended by two folds of this membrane, the phrenicolienal ligament and the gastrolienal ligament. The friable, fragile nature of the splenic tissue, together with its mode of suspension explain the frequency with which the spleen is ruptured by indirect force despite its seemingly protected position. Also, since the large vessels supplying the spleen run through the gastrolienal ligament, it is easily seen that a tear of this ligament would result in grave hemorrhage.

The splenic artery, next to the renal arteries, is the largest vessel supplying any of the abdominal viscera. It is the largest branch of the celiac axis, and is out of all proportion to the size of the organ it supplies. The artery divides into a series of branching networks which finally allow the blood to percolate through the interstices of the reticulo-endothelial tissue. Thus it may be seen that, for the purposes of our consideration, the spleen may be thought of as an organ made up largely of blood vessels and spaces for blood within the loose pulp. A fracture in its substance may be considered as a tear in one of the most vascular tissues in the body.

The nerve supply to the spleen comes from the celiac plexus. Stimulation of these autonomic fibers leads to contraction of the smooth muscle fibers within the capsule and trabeculae.

The physiology of the spleen is still not completely understood. However, three well recognized functions may be stated, namely, the destruction of formed blood elements, the storage of blood, and the production of lymphocytes in the Malpighian corpuscles. It is with the second of these functions that we are particularly concerned in this discussion.

Stuckey, as early as 1742, stated that the spleen acted as a blood reservoir. But it was not until the work of Barcroft[3] was published (1922 through 1936) that the dynamics of this function were understood. Through these studies as well as the work of others, Holman,[24] Hargis and Mann,[32] it is now known that the spleen contracts in asphyxia, exercise, hemorrhage and emotional excitement. Doan, Curtis and Wiseman[16] actually demonstrated the contraction of the human spleen by injecting adrenalin into the splenic artery at operation and noting its diminution in size. In effect, this actually results in a largely cellular autotransfusion. This procedure together with massage is used routinely by one of us (G. M. C.) in the course of splenectomies for pathologic spleens.

Still more important was the demonstration by Barcroft and Nisimaru[3] that the spleen undergoes constant, spontaneous, rhythmic contractions. These have a duration of from twenty-five to fifty seconds and are reflected as low undulations in blood pressure tracings. The practical import to our later discussion of treatment is obvious. The surgeon who contents himself with suture or packing and is aware of these contractions, can hardly conclude that he has used the safest method of arresting splenic hemorrhage.

ETIOLOGY

The cause of traumatic rupture of the spleen is some form of direct or indirect physical violence. Less frequently the rupture is due to a penetrating wound.

Many pathological conditions cause enlargement of the spleen. That these spleens are subject to traumatic rupture is revealed in the reports of Banerjee[2] and Henderson.[22] Some authors suggest that the pathological changes which these spleens have undergone make them even more susceptible to traumatism and consequent rupture.

That the normal human spleen may rupture spontaneously has been suggested. Susman[41] collected seven and Zuckerman and

Jacoby[49] collected twenty cases purported to be of this type. How-
ever, a more critical review of the available literature casts some
doubt on the probability of such an occurrence. It is more likely that
these were instances of delayed rupture of previously traumatized
spleens; the history of trauma being overlooked or even forgotten.
We are hesitant to conclude that spontaneous rupture of a normal
spleen can occur.

TABLE I

SEX INCIDENCE OF TRAUMATIC RUPTURE OF SPLEEN

Sex	Incidence
Female	2
Male .	20
	—
Total	22

TABLE II

INCIDENCE OF TRAUMATIC RUPTURE OF SPLEEN BY AGE GROUPS

Age Group	Incidence
1–10	5
11–20	7
21–30	4
31–40	2
41–50	2
51–60	1
61–70	1
	—
Total	22

TABLE III

VARIOUS TYPES OF ACCIDENT PRODUCING TRAUMATIC RUPTURE OF THE SPLEEN

Type of Injury	Incidence
Athletic	1
Automobile	11
Bicycle	1
Bullet wound	2
Fall	5
Industrial	2
	—
Total	22

Traumatic rupture of the spleen occurs more frequently in males
(Table I); only 9.1 per cent of our patients were women. Likewise age
is an important influencing factor. Young individuals are far more
often affected than older people (Table II); 81 per cent of our cases
occurred before the age of forty years. This may readily be explained
by the greater activity of youth.

The manner in which splenic injury is ordinarily sustained is
significant. In practically all recent reports, including our own,
automobile accidents have accounted for the greatest number. (Table
III.) In our series, automobile accidents accounted for eleven cases, or

half of the entire group. It is interesting to glance back at the excellent earlier discussion of the subject by Conners,[13] in 1921, and find that the automobile's predecessor, the horse and wagon, was then the most frequent cause of the injury. A bit of irony is to be found in the report of Dretzka,[18] from Detroit, the capitol of the automobile world, where not the automobile, but gunshot and stab wounds accounted for the largest number of injuries.

Clear-cut industrial accidents account for but 9 per cent of our cases. This suggests that the long campaign for industrial safeguards is having some effect. Falls account for the largest number of the injuries in children.

It is interesting to note that in Gordon-Taylor's discussion[21] of war injuries occurring in England, a new and more bizarre cause of traumatic rupture of the spleen is mentioned. This is rupture due to blast injury. In this respect the spleen is the most frequently affected abdominal organ.

Considering at length the available reports of war injuries, we find that in military as in civilian surgery, the spleen is the most frequently affected organ in subcutaneous injury to the abdominal viscera. The frequent involvement of the spleen in civilian surgery is shown by the reports of Butler and Birnbaum,[11] Bronaugh[8] and Wright and Prigot[48] who find the spleen involved in 30, 33 and 47 per cent, respectively, of all subcutaneous abdominal injuries. Rupture of the spleen is even more frequent in occurrence than rupture of the liver or kidney which follow it in frequency.

PATHOLOGY

From examination of the spleens removed at operation and during necropsy, it is found that traumatically ruptured spleens can usually be classed in one of five categories. These more or less arbitrary groups are illustrated in Figures 1, 2, 3 and 4. Each type corresponds with a different clinical picture. This knowledge is of aid in both diagnosis and treatment.

Type I is a complete fragmentation of the spleen into two or more parts, or a complete tearing of the spleen from its pedicle. This results in immediate massive hemorrhage and in many instances in sudden death. Figure 4 illustrates a spleen of this type, removed in Case 9 of our series. The patient was kicked in the back while playing ball. He rapidly went into shock. He was operated upon within two hours of the injury and made a complete recovery. (Fig. 1.)

Type II represents a large tear at or near the hilus. This gives rise to the rapid onset of grave hemorrhage and like Type I demands the most rapid surgical intervention. (Fig. 2.)

TYPE I   TYPE II.
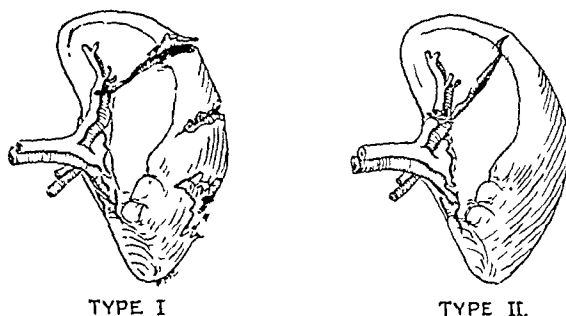
Fig. 1. Diagrammatic representation of Types I and II of splenic rupture, both leading to rapidly progressive hemorrhage. Variations of Type I are a complete severance of the pedicle and a complete disruption of the spleen (Fig. 4).
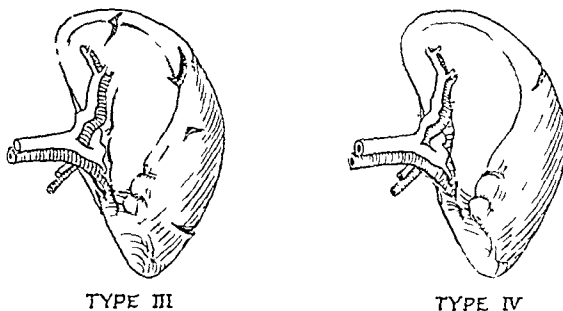
TYPE III   TYPE IV

Fig. 2. Diagrammatic representation of Types III and IV of splenic rupture. Type IV is the lesion commonly responsible for the development of a perisplenic hematoma.

Type III is characterized by one large or multiple smaller tears about the periphery of the spleen. This type leads to slow but steady development of the signs of intra-abdominal hemorrhage. (Fig. 2.)

Type IV presents a solitary small tear at the periphery. (Fig. 2.)

Type V is characterized by subcapsular hematoma. (Fig. 3.)

The last two types are those particularly responsible for the development of the syndrome of *delayed hemorrhage* which will be discussed under symptomatology.

One further pathologic consideration is of importance. This is whether or not spontaneous cure of a splenic hematoma is possible. If such is possible, there is some justification for those who believe that this lesion should be treated conservatively. R. C. Webb[45]

quotes J. S. McCartney as reviewing 25,000 necropsies at the University of Minnesota. In this large group he found no instance of a healed splenic hematoma. It is our conclusion that such a spontaneous cure rarely if ever occurs.
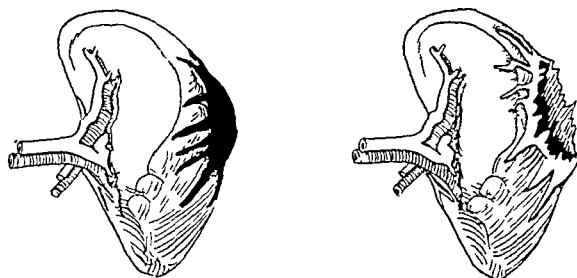
TYPE V.

FIG. 3. Diagrammatic representation of Type V splenic rupture. This presents two stages in the evolution of a subcapsular hematoma, the last representing its rupture.

FIG. 4. Photograph of a Type I splenic rupture. This spleen was removed from Case 9 of our series.

SYMPTOMATOLOGY

There is no one pathognomonic sign or symptom of traumatic rupture of the spleen. In almost every textbook of surgery Kehr's sign and Ballance's sign are prominently mentioned as diagnostic.[14] This has not been our experience, particularly with reference to Kehr's sign, which consists of referred pain in the left shoulder from irritation of the left diaphragm. A consideration of the literature

reveals that, while many writers mention it, few actually noted its occurrence in their own cases. Consideration of the pathologic findings in splenic rupture would indicate that it should be particularly found in instances of delayed hemorrhage. Ballance's sign is more frequently encountered. It consists of dullness to percussion in the left upper quadrant, left flank and even in the right flank. On the shifting of position, only the dullness in the right flank disappears.

TABLE IV

INCIDENCE OF COMMON SIGNS AND SYMPTOMS OF SPLENIC RUPTURE

In Order of Frequency

Generalized abdominal rigidity	13
Shock	12
Pain in left upper quadrant	12
Generalized abdominal pain	11
Rigidity in left upper quadrant	4
Initial unconsciousness	4
Pain on respiration	4
Dullness in left upper quadrant	4
Vomiting	3
Dullness in both flanks	2
Pain in left lumbar area	1

Table IV presents the incidence of the common signs and symptoms. The most frequently encountered findings are abdominal pain and rigidity, either generalized or localized to the left upper quadrant. Shock is another frequent finding, occurring in over half of our cases; however, it is not always present at the time the surgeon is called upon to give his judgment. We have noticed an initial shock which disappeared spontaneously. This has recurred in certain instances as the hemorrhage progressed. In other patients shock develops only as the effects of blood loss become otherwise evident. Initial loss of consciousness and vomiting were two of the less frequently encountered findings.

No discussion of traumatic rupture of the spleen would be complete without consideration of the syndrome of *delayed hemorrhage.* This was first described by A. H. McIndoe.[34] In 1932, McIndoe reported forty-five cases. Since then we have found thirteen more cases reported in the literature.[6,7,8,15,17,20,25,38,40,45,46,48] To this list we are able to add two more of our own.

This definite clinical picture merits more complete understanding, otherwise many a case will be overlooked. It is, in all probability, more common than the literature indicates. The syndrome may be described in the following manner: After an injury to the back, lumbar area, or abdomen, which frequently is not of great severity, the

patient suffers more or less pain, some nausea and perhaps even mild shock. After several hours these symptoms disappear completely, or at most, leave only a vague upper-abdominal discomfort. The patient then enters the so-called latent period and is able to be up and about.

TABLE V*

TIME ELAPSED BETWEEN INJURY AND ONSET OF SYMPTOMS

Time Interval	No. of Cases
Immediate	15
1–10 hours	3
11–48 hours	2
Delayed hemorrhage	2
Total	22

* This table shows the time interval elapsing between injury and the onset of symptoms leading to a diagnosis of ruptured spleen. The last two instances of delayed hemorrhage occurred eight days and five weeks, respectively, after injury.

The minimum length of the latent period should be forty-eight hours, as defined by McIndoe,[34] to differentiate it clearly from simple, slowly progressive hemorrhage which may take more than twenty-four hours to produce signs and symptoms. (Table v.) Also during the latent period the patient should be practically symptom-free. Latent periods as long as six months are reported in the literature. The latent period, after forty-eight hours or more, is suddenly terminated by the return of severe symptoms of intense abdominal pain and shock produced by the sudden release of a large amount of blood into the peritoneal cavity. This may occur so long after the unappreciated initial injury that the accident is even forgotten and the case considered a spontaneous rupture. During the latent period warning signals do exist. There will usually be dull pain on pressure over the left upper quadrant, a rapid pulse and a leukocytosis.

The entire syndrome is better understood when the underlying pathologic physiology is made clear. Types iv and v of splenic injury particularly predispose to its development. (Figs. 2 and 3.) Thus, a relatively moderate blow may be followed by a small subcapsular hematoma with slight maceration of the adjacent splenic pulp. The spleen appears unable to heal this type of injury. Following this, constant oozing into the subcapsular hematoma gradually causes it to expand, stripping the capsule from the spleen and further macerating the pulp. The hematoma swells to considerable size. Finally, a slight movement or strain is sufficient to cause it to rupture, releasing blood into the peritoneal cavity. A similar picture results when a small tear permits slow hemorrhage to occur. Sufficient time is thus allowed for the omentum, colon and stomach to wall off an area

about the spleen. A perisplenic hematoma is thus formed which may reach great size before the latent period is terminated by some slight strain or sudden movement sufficient to break down the barrier of adhesions.

Our two cases of delayed hemorrhage follow:

CASE 5.   The patient, V. La F., a student at The Ohio State University, was admitted to the University Hospital shortly after an automobile accident, complaining of intense abdominal pain. Immediately after the accident he lost consciousness, but did not appear to be in shock at the time of admission.

Examination at the time of admission revealed: Temperature 98°F.; pulse 80; respirations 22; blood pressure 120/80. Respiratory movement of the left chest was decreased and left lower chest pain was experienced on inspiration. The abdomen showed marked generalized rigidity with marked tenderness in both upper quadrants. No masses were felt. The admission erythrocyte count was 5.06; the leucocyte count was 17,280.

The patient was admitted and placed under observation. By the next morning he felt well except for some residual soreness in the left upper quadrant. He was, however, kept in the hospital and in bed. Five days after admission his erythrocyte count was 3.42 and his leucocyte count was 21,900.

On the eighth hospital day the patient felt well and was allowed to sit in a chair. That evening almost immediately after returning to bed, he experienced severe abdominal pain and went into shock. His erythrocyte count at this time was 2.59 and his leucocyte count was 35,200. He was given a blood transfusion and taken to the operating room. At operation a ruptured, subcapsular splenic hematoma was found. This was treated by tamponade and suture. The patient made a complete recovery.

CASE 15.   J. B., an eighteen-year old white youth, was admitted to the University Hospital complaining of severe abdominal pain, nausea and vomiting. Five weeks prior to admission he was in an explosion in which he received a blow to his left upper quadrant. He was observed in another hospital for two days, but upon revealing no signs of severe injury was discharged. He thereupon resumed his normal activity. Twelve hours prior to his second admission he developed severe generalized abdominal pain of a cramp-like nature. He became very weak, was nauseated and vomited.

Examination on admission revealed: Temperature 100°F.; pulse 110; respirations 20; blood pressure 90/50. The patient lay quietly in bed with both thighs flexed, and in great pain. The lungs were clear; the heart was normal. The abdomen revealed generalized rigidity and tenderness. The patient's erythrocyte count was 3.90 and the leucocyte count was 22,400.

Twelve hours after admission the patient was operated upon. A perisplenic hematoma was found. The spleen had a small peripheral laceration.

This was sutured and the splenic fossa packed. The patient made an uneventful recovery.

Before considering the diagnosis of splenic rupture, the subject of associated lesions should be considered. Wright and Prigot[48] found severe associated injuries in 50 per cent of their cases. In their series they encountered the following lesions, in descending order of frequency: Fractured ribs, fractures of the extremities, lacerated kidney, hemathorax with lacerated lung, cerebral complications, ruptured liver, ruptured urinary bladder and ruptured diaphragm.

TABLE VI*
ASSOCIATED INJURIES AND FATALITY

Type of Injury	Incidence	Fatality
Fractured ribs . .	5	None
Hemothorax . .	3	1
Fracture of extremities	3	None
Contusion of pancreas	1	None
Ruptured kidney .	2	2
Retroperitoneal hemorrhage	1	1
Ruptured liver .	2	1

* The associated pathologic lesions listed in order of frequency found in our group of 22 cases of splenic rupture. The effect of each complicating lesion on the outcome is indicated in the column at the right.

In Table VI the type and frequency of the associated lesions in our own series is recorded. In ten patients, or 45 per cent of the group, other injuries were present. The table also shows the effect of the associated damage on the outcome of the case. Undoubtedly, the presence of other injuries portends a poorer prognosis. However, the authors wish to make this point: Too often the associated condition, which may be more obvious but less serious, distracts the surgeon's attention. Only belatedly does he realize that the hemorrhage from a ruptured spleen is killing his patient. By the time this realization comes it may be too late.

An illustrative example is our Case 16, admitted in shock, with a fracture of the neck of the right femur and a fracture of the shaft of the left femur. The fractures were treated correctly, yet the patient died of hemorrhage from a ruptured spleen.

One further point should be made in connection with severe associated injuries. In reviewing our series we believe that too often when confronted with multiple severe lesions, the surgeon hesitates,

or is overwhelmed by the magnitude of the task before him. He temporizes, delays while treating the shock, and with the delay follows loss of the patient. Cases with trauma to other organs call for the most energetic intervention of all and even delay in treating the shock may result fatally.

Two cases in our series are again illustrative. Both had ruptured spleens and ruptured livers. (Table VI.) In one, operation was delayed thirty-six hours. The patient died. In the other operation was performed immediately after admission, while the patient was still in shock, with a blood pressure of 70/40. A splenectomy was accomplished and the liver sutured. The patient made an uneventful convalescence and was discharged on the twentieth postoperative day.

DIAGNOSIS

The diagnosis of traumatic rupture of the normal spleen may be very simple in the typical uncomplicated case, or extremely difficult when associated with additional injuries. The associated injuries may be so severe as to mask or even overshadow the symptoms of ruptured spleen. One of the cardinal symptoms, found constantly in reviewed cases, is that of abdominal pain either generalized or confined to the left side of the abdomen. This pain is usually very severe and may be out of all proportion to the suspected severity of the injury. History elicits some type of external violence. The patient presents severe progressive shock and may give a story of periods of unconsciousness. Breathing is usually thoracic in character, with respirations rapid and shallow. Ballance's sign may or may not be positive. On examination the surgeon is impressed by the rigidity of the abdomen which increases with the shock and the seriousness of the injury. The temperature early is subnormal, the pulse rapid and of poor quality, depending upon the degree of hemorrhage and shock.

In addition to the above clinical findings certain laboratory procedures may be done quickly and are of definite help. The leucocyte count in 80 per cent of the cases will be elevated above 10,000 and on repeat counts will be found to be rising. The erythrocyte count and hemoglobin may show a slight increase above normal at first (Table VII) but on repeated counts will be found to be falling rapidly. The falling drop method of Scudder for determining early hemoconcentration in incipient shock can be run quickly and will prove most helpful in making an early diagnosis.

Webb[45] has stressed the importance of a roentgenogram of the abdomen. This will frequently show elevation of the left diaphragm. The authors agree with this view completely. The procedure takes but little time and can be most helpful, particularly in cases of delayed hemorrhage.

TABLE VII

BLOOD AND URINARY FINDINGS ON ADMISSION IN OUR TWENTY-TWO CASES
OF SPLENIC RUPTURE

	No of Cases
Red Blood Cells	
Above 4 00	7
Above 3.00	9
Less than 3.00	3
White Blood Cells	
Above 20,000	7
Above 10,000	9
Less than 10,000	2
Red Blood Cells in Urine	5

Wright and Prigot[48] stress the use of paracentesis to determine abdominal hemorrhage. This is undoubtedly of value in certain instances, but we do not believe that its routine use, as suggested by them, is necessary or justified.

Burke and Madigan[10] suggest the intravenous injection of thoratrast. This is an ingenious method and in a limited number of obscure cases may be most helpful. The reticulo-endothelial elements of the spleen phagocytize particles of the contrast medium. But a delay of at least two hours is necessary to interpret the test.

The conditions from which ruptured spleen must be differentiated are mainly acute abdominal emergencies which require prompt surgical intervention. Perforated peptic ulcer, mesenteric thrombosis, ruptured kidney or liver, ruptured tubal pregnancy may all be mentioned. Of all these, rupture of the left kidney is the most likely to cause confusion. Whenever this enters as a possibility, analysis of the urine and an intravenous pyelogram can be done quickly and should serve to make the differential diagnosis.

We wish to stress the importance of early diagnosis. Fifty-one per cent of patients with ruptured spleens die within one hour following injury. We realize that but few of these cases may have the opportunity for adequate treatment, due to the severity of their injury and the rapidity of its course. The mortality rate in the remaining 49 per cent may be lowered by prompt diagnosis and adequate treatment. All patients with a history of abdominal injury should be carefully examined, re-examined and observed, bearing in mind the probabil-

ity of injury to the spleen, and the seriousness of the situation should this exist. The surgeon should realize that an error in diagnosis or a tendency to minimize the situation may be fatal to the patient.

TABLE VIII

ADMISSION AND PREOPERATIVE DIAGNOSES IN OUR SERIES

Ruptured spleen	6
Ruptured intra-abdominal viscus	4
Intra-abdominal hemorrhage	4
Possible ruptured spleen	2
No diagnosis	2
Fractured ribs—Intra-abdominal injury	1
Acute suppurative appendicitis	1
Laceration of lung	1
Coronary occlusion	1
Total	22

Finally, the importance of making the correct diagnosis preoperatively cannot be overemphasized. Table VIII lists the preoperative or admission diagnoses in our series. In only six cases, or 27 per cent of the series was the correct diagnosis made preoperatively. Of these only one died, giving a mortality of 16 per cent. Of the remaining sixteen cases incorrectly diagnosed, or undiagnosed, six died resulting in a mortality of over 37 per cent.

TREATMENT

A brief backward glance at the early attempts at surgery for traumatic rupture of the spleen is interesting. The first successful splenectomy for splenic rupture was performed in 1892.[5] By 1900, Bessel-Hagen[4] reported thirty-seven patients operated upon with twenty recoveries or a mortality of 46 per cent. Lotsch,[30] in 1908, reported 138 operative cases with a mortality of 37 per cent. With general improvement in surgical procedures and a more widespread awareness of the occurrence of ruptured spleen, this rate has gradually lowered. By 1922, Buxton,[12] reviewing the cases reported up to that time, found a mortality rate of 28.8 per cent of all patients operated upon.

The treatment of traumatic rupture of the spleen or of rupture with delayed hemorrhage is surgical. Conservatism has but small place in the treatment of ruptured spleens. Wright and Prigot[48] record the mortality in conservatively treated cases, in those refusing treatment, or in those undiagnosed, as from 90 to 100 per cent. They also state that 51 per cent of patients with a ruptured spleen die within

one hour following their accident. We may conclude from these statistics that *surgical treatment must be prompt and energetic.*

Patients presenting shock should be treated by shock combatting methods such as intravenous fluids, plasma, or by slow drip blood transfusion *during* surgery. It is impossible to expect to alleviate shock in a patient who is hemorrhaging from a large tear in the spleen. If operation is delayed until the patient recovers from shock, but few ruptured spleens will come to operation. We cannot stress too strongly that shock is not to be considered a contraindication to surgery in this condition.

Each patient should be treated individually. The choice of the anesthetic agent, depending upon facilities at hand, should be made with regard for the safety of and suitability for each particular case. Many patients are started under local and supplemented with inhalation anesthesia.

On opening the abdomen, in nearly all cases of ruptured spleen, a large amount of free blood is found. This blood should be aspirated into a sterile container, filtered and then reinjected into the patient's vein. Autotransfusion[26,28] originally advocated in the literature by Kraft, in 1931, is but rarely mentioned yet will be found to be lifesaving in many cases. No ill effects occur since the blood is not contaminated. Post-transfusion reactions do not occur as they sometimes do in using donor blood, while the free blood in the abdominal cavity affords a means of prompt transfusion to each case. One of us (W. D. N.) has used this method most successfully.

The ruptured spleen should be quickly inspected and frequently may be grasped with the hand to control bleeding before clamping the splenic pedicle. From careful consideration of our own series and a review of many other cases, we have concluded that suture or tamponade has no place in the treatment of the ruptured spleen. *Any fractured spleen should be removed.* When the splenic vessels are suitably ligated any danger from hemorrhage has been obviated. Due to the consistency of the organ it is most difficult to suture the spleen satisfactorily. Tamponades may control hemorrhage while *in situ,* but their removal is frequently followed by recurrent or even by delayed hemorrhage. We have no assurance that a sutured spleen will heal satisfactorily or that delayed hemorrhage will not occur.

The few who advocate suture and packing are at variance with the majority opinion on the subject. Dretzka[18] writing of civilian practice and Gordon-Taylor[21] reporting on war injuries to the spleen, both hold that suture is satisfactory for gunshot and penetrating

wounds. However, Jolly[27] also writing of war surgery, is definitely against such a procedure.

The operation should be done as quickly as possible with adherence to the fundamental principles of surgery. Hemostasis must be

TABLE IX*

TREATMENT BY SPLENECTOMY IN RELATION TO MORBIDITY AND MORTALITY

Case No.	Associated Lesions	Mortality	Stay in Hospital—Days
1	Contusion of pancreas	Recovered	35
3	Pneumonia	Recovered	89
4	Hemothorax	Recovered	17
7	Fractured extremities	Recovered	12
8	None	Recovered	21
9	None	Recovered	30
11	None	Recovered	14
12	Fractured ribs	Recovered	22
13	None	Recovered	8
18	Ruptured liver	Recovered	24
22	Ruptured diaphragm and lung	Died	1
11	7	1	27

* This table lists the results in eleven cases treated by splenectomy. The associated lesions are included to indicate the severity of the injury. Morbidity is measured by the length of stay in the hospital.

secured; a rapid but careful search for additional injuries such as perforation of the stomach or bowel in penetrating wounds; and rupture of the diaphragm, kidney, or liver, in crushing injuries must be made. Wounds not contaminated are closed without drainage.

Postoperatively, these patients are treated as any other surgical patient in shock or after hemorrhage. Blood transfusions should be given. We have found repeated small transfusions given frequently of more benefit than the same amount of blood given as a larger transfusion. Fluids by vein and clysis, and blood plasma also have their place in postoperative care. Morphine should be used freely to produce rest and relaxation.

The data in Tables IX, X, XI, and XII summarize our findings in regard to treatment. Table IX reveals that eleven of our twenty-two patients were treated by splenectomy. In this group there was one death. This is a mortality of 9 per cent. The patient who died was long delayed in securing medical attention and was practically *in extremis* when operated upon. Table X shows that four patients were treated by suture or packing. Of these none died. The mortality

rate for the fifteen operated cases is thus 6.6 per cent. This is an exceptionally low figure. The average reported in the literature is about 25 per cent. Yet, when this is compared with the mortality

TABLE X*

TREATMENT BY SUTURE AND PACKING IN RELATION TO MORBIDITY AND MORTALITY

Case No.	Associated Lesions	Mortality	Stay in Hospital—Days
5	None	Recovered	33
14	None	Recovered	33
15	None	Recovered	22
19	None	Recovered	62
4	0	0	37

* This table presents the results of more conservative therapy in the management of four cases of splenic rupture. There were no severe associated lesions.

TABLE XI*

MORBIDITY AND MORTALITY WHEN NO TREATMENT IS ATTEMPTED

Case No.	Associated Lesions	Mortality	Stay in Hospital—Days
2	Ruptured kidney	Died	1
6	None	Died	2
10	None	Recovered	36
16	Ruptured liver	Died	2
17	Ruptured kidney	Died	1
20	Hemothorax	Died	2
21	Rheumatic heart	Died	11
7	5	6	8

* This table shows the results obtained in 7 cases of splenic rupture when no treatment other than observation or medical management was attempted.

rate of 85 per cent in the untreated group, it is convincing that conservative or nonoperative treatment has little if any place in the management of traumatic rupture of the spleen.

A comparison of Table IX and X as summarized in Table XII presents the difference in morbidity between those patients treated by splenectomy and those treated by suture or packing. As measured by length of hospital stay, the patients treated by splenectomy showed a lower morbidity than those treated by other operative measures—twenty-seven days as compared to thirty-seven days.

The question of spontaneous cure usually arises in any discussion of treatment. Some surgeon can usually be found who will recite his

case of ruptured spleen in which the patient made a spontaneous recovery. To deny categorically that such is possible would be imprudent. We even include one such case in our group.

TABLE XII

SUMMARY OF RESULTS OBTAINED BY THE VARIOUS TYPES OF TREATMENT IN RELATION TO MORBIDITY AND MORTALITY

Treatment	No. of Cases	Mortality	Stay in Hospital—Days
Splenectomy	11	1	27
Suture and packing	4	0	37
No operation	7	6	8

CASE 10. H. S., a twenty-eight-year old white male, was admitted to the hospital complaining of pain in the left upper quadrant after being struck by an automobile. He did not lose consciousness but immediately after injury noted pain in the left lower chest and left upper abdomen.

Examination revealed: Temperature 101°F.; pulse 90; respirations 48; blood pressure 106/54. There was marked tenderness in the left upper quadrant and flank and dullness to percussion in the left flank. Admission erythrocyte count was 4.69; the leucocyte count was 32,100.

Immediately after admission the patient went into shock. His blood pressure dropped to 80/50. He was treated conservatively and made rapid improvement. On his eleventh hospital day he was discharged. On the way home, while still in a taxi, he again suffered severe abdominal pain and collapsed. He was immediately returned to the hospital, arriving in shock with a blood pressure of 84/60. The signs and symptoms on examination were identical with those of his previous admission. Again he was treated conservatively and gradually improved. He was finally discharged on his twenty-fifth hospital day.

This case illustrates well the matter under discussion. In the first place, if operation is not performed, the surgeon cannot be sure that the patient had a ruptured spleen. Second, having once grasped the full implication of delayed hemorrhage, the surgeon cannot quite be sure but that the patient will have a recurrence of the bleeding at a later date. If, in the case presented above, the patient had gone to another surgeon at a different hospital after his first discharge, the original surgeon would still be talking about his case of spontaneous recovery. Even at present there is no certainty but that two days or two months after his second discharge, the patient will not again have a delayed hemorrhage.

To summarize then, we conclude that the treatment of traumatic rupture of the spleen is always surgical and that the operation of choice is splenectomy.

COMPLICATIONS

The most commonly mentioned postoperative complication is intestinal obstruction, due either to paralytic ileus or adhesions from trauma to the intestines and mesentery. Three such instances occurred in our group. Disruption and infection of the wound may occur as a result of injury to the tail of the pancreas, or to undue haste in wound closure. One of our patients suffered from this complication. Postoperative lung complications are frequent because of associated injury to the chest. In our series there were three postoperative pneumonias in addition to the hemothoraces already listed. These and other complications must be recognized early and suitably treated.

POSTOPERATIVE RESULTS OF SPLENECTOMY FOR TRAUMATIC RUPTURE OF THE SPLEEN

It is significant to note that but few cases have been carefully studied postoperatively after having had a splenectomy for splenic rupture. Yet here is a most fertile field in which to add to our all too hazy knowledge of the normal function of the spleen. Many reports have been written on the postoperative results of splenectomy for pathologic conditions of the spleen,[31,45,47] but here in a group of vigorous, healthy individuals suddenly deprived of a normal spleen, but little investigation of real value has been accomplished.

The material embodied in this section may be considered as a preliminary report of work in progress by one of us (L. C. R.).

In 1924, Pfeiffer and Smyth[37] made one of the few reports specifically dealing with the late results of splenectomy for traumatic rupture of the spleen. Before that Hitzrob,[23] in 1918, published a paper on "The Effect of Splenectomy on Normal Individuals and in Certain Pathological Conditions."

The resultant effects which these authors and others generally attribute to such splenectomies may be listed as follows: (1) An anemia which gradually returns to normal in from three to four months; (2) an increased resistance to destruction of the red cells; (3) an increased output of iron in the stools; (4) an increased total blood cholesterin; (5) no change in opsonins and agglutinins; (6) pronounced enlargement of the superficial lymph glands; (7) ease of

fatigability, and (8) lowered number of blood platelets, considering 600,000 as normal.

These results are based on four cases investigated at periods

TABLE XIII

LATE POSTOPERATIVE FOLLOW-UP OF FOUR CASES OF TRAUMATIC RUPTURE OF THE SPLEEN
TREATED BY SPLENECTOMY

| Case No. | Age at Splenectomy Yrs. | Interval Since Splenectomy Yrs. | Hematology | | | | | Growth | Increased Incidence of Infection |
			Hemoglobin	Red Blood Cells	White Blood Cells	Platelets	Differential		
1	14	2	14 5	4.90	7,200	800,000	Normal	Adequate	None
12	8	7	14	4 00	7,500	848,000	Normal	Adequate	None
9	20	3	14 5	4 90	6,250	690,000	Normal	Adequate	None
11	12	3	13.5	4 25	6,500	700,000	Normal	Adequate	None

ranging from one year and nine months to four years postoperatively. Other observers have attributed a failure of proper growth and a decreased resistance to infection to the removal of a normal spleen.

Our studies on this subject are based on six patients who received splenectomies for splenic rupture. The period postoperatively ranges from two to seven years. Four have been partially investigated, while two have only had a preliminary interview. We are most fortunate in having the assistance of the hematologic laboratory of Drs. C. A. Doan and B. K. Wiseman in carrying out this work.

Table XIII gives certain of our findings in our first four cases. The columns headed "Growth" and "Infection" indicate that according to clinical tests there was no deficiency of the one or increased evidence of the other. Another fortunate circumstance, as shown in the table, is that all these cases were adolescent at the time of splenectomy.

Specifically our investigation thus far shows that all four patients have a normal hematologic picture. None showed either anemia or thrombopenia. All four showed normal growth. None showed any increased incidence of infection. None of the four had any appreciable enlargement of the superficial lymph glands. All four had normal basal metabolic rates ranging from minus 2 to plus 8. In all the plasma iron was within normal limits. None admitted any decrease in energy or ease of fatigability. All were actively engaged in sports and extracurricular scholastic activities.

Three, however, have shown one significant finding. When their blood volumes were investigated they were found to be normal. How-

ever, after the injection of adrenalin their blood volume did not increase as does that of the normal individual. It remained remarkably constant. Furthermore their blood pressures showed only a 6 to 8 millimeter of mercury increase. They did, however, show an increase in pulse rate. The fourth case has not yet been thus investigated. Therefore, to the question, "What is the effect of splenectomy on a normal individual?", we must perforce answer, "We have found none." So far as health and growth are concerned, the spleen appears to be an unnecessary organ.

One other late result of traumatic rupture of the spleen should be mentioned. In 1892, Albrecht[1] reported a case which at autopsy was found to have no primary spleen but numerous small growths of splenic tissue on the greater omentum, the diaphragm and in the pouch of Douglas. There was no history of trauma or of splenectomy. These growths may have been multiple accessory or ectopic spleens which have been found in a high percentage of neciopsies. Albrecht, however, reported them as splenic autotransplants.

In 1910, von Kuttner reported a case, necropsied four years after splenectomy, in which from eighty to one hundred splenic growths were found scattered throughout the abdomen. These could hardly have been accessory spleens. Since then Foltin,[19] von Stubenrauch,[44] Oltman,[44] Lee,[29] Kupperman,[42] Shaw and Shaft[39] in Europe; and Buckbinder and Lipkoff[9] in this country, have each reported one case of splenic autotransplants. Buchbinder and Lipkoff have suggested the name *splenosis* for this condition.

In 1920, Marine and Manley[33] successfully made autotransplants of splenic tissue to the abdominal muscles of rabbits. In 1936, Perla[36] produced peritoneal splenic autotransplants in rats.

In our own experimental work we have produced peritoneal splenic autotransplants in three out of six rabbits. These were not recognizable earlier than six months after implantation. Figures 5, 6 and 7 present the gross appearance and microphotographs of the transplants in one such experimental splenosis. Our technic has been to splenectomize the rabbit under conditions of strict asepsis. The spleen is then aseptically macerated to the consistency of a thick paste. Small portions of this, no larger than a pinhead, are then seeded throughout the abdomen.

From this experimental study we conclude that splenosis is a rare but definite and serious late complication of splenic rupture. The seriousness lies in the fact that the splenic tissue may be simultane-

ously implanted on two adjoining loops of bowel. This may, in time, provide the basis for an intestinal obstruction. One such case has been reported. Another untoward result of splenosis could conceiv-
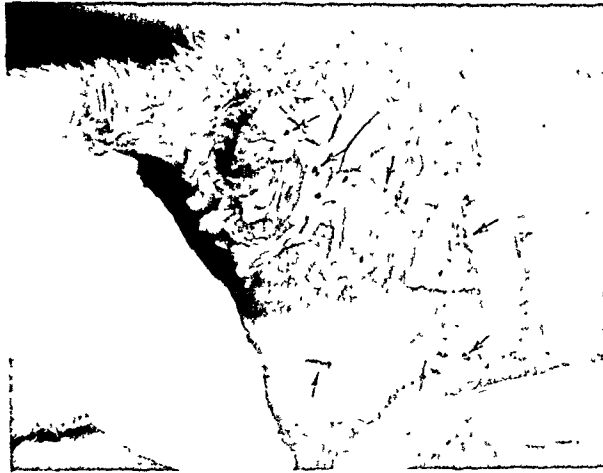

FIG. 5. A photograph of the intestine, mesentery, and parietal peritoneum of a rabbit showing numerous areas of autoplastic splenic transplants.

FIG. 6. Microphotograph, low magnification, showing a portion of a splenic autotransplant attached to the serosa of the intestine of a rabbit.

FIG. 7. Microphotograph, high magnification, of the same specimen as Figure 6, showing cellular detail of splenic autotransplant.

ably be the development of an abnormal hemoclastic activity by the transplanted splenic tissue.

CONCLUSIONS

Traumatic rupture of the normal human spleen is not as rare as is commonly thought. Its incidence, both real and apparent, is increasing in civilian life and is high in war injuries. The spleen is the most frequently injured of all the abdominal viscera. The types of injury most frequently causing this lesion, aside from war injuries, are automobile accidents and falls. Five types of pathologic rupture are recognized, each leading to a definite clinical picture. Of these the syndrome of delayed hemorrhage is the most frequently overlooked.

Associated severe lesions occur in about 50 per cent of the cases. These materially increase the likelihood of a fatal outcome and may call for rapid surgical intervention.

There are no pathognomonic signs or symptoms of splenic rupture. Generalized abdominal rigidity and tenderness, or the same more localized to the left upper quadrant are encountered in the majority of cases. Shock is also most frequently noted. These clinical findings together with certain simple laboratory tests enable the surgeon to make the diagnosis with certainty. The laboratory tests of greatest value are: repeated determinations of the hemoglobin and erythrocyte count, the Scudder falling drop test for hemoconcentration and a flat plate of the abdomen showing an elevated left diaphragm.

The treatment of traumatic rupture of the spleen is always surgical. Splenectomy is the operation of choice. Operation should not be delayed in an effort to combat shock. This should be treated while the operation is in progress. Autotransfusions are recommended.

Ultimate ill effects subsequent to absence of the spleen after splenectomy for splenic rupture are negligible. We have been able thus far to determine no ill effects. Splenosis or the occurrence of splenic autotransplants within the peritoneal cavity is a real but rare late complication.

REFERENCES

1. ALBRECHT. Ein Fall von sehr zahlreichen uber das ganze Peritoneum versprengten Nebenmilzen. *Beitr. z. path. Anat.*, 20: 513, 1896.
2. BANERJEE, P. Surgery of the ruptured spleen. *Indian Med. Gaz.*, 70: 273, 1935.
3. BARCROFT, J. and associates. Observations upon the effect of high altitude on the physiological processes of the human body. On the relation of external temperature to blood volume. *Phil. Tr. Roy. Soc.*, B. 211: 351, 1922. Observations on the taking up of carbon monoxide by the hemoglobin in the spleen. *J. Physiol.*, 58: 138, 1923. Contribution to the physiology of the spleen. *Ibid.*, 60: 443, 1925. The influence of the spleen in carbon monoxide poisoning. *Ibid.*, p. 79. Rhythmical contractions of

the spleen. *Ibid.*, 74: 294, 1932. Cause of rhythmical contractions of the spleen. *Ibid.*, p. 299. Undulatory changes in blood pressure. *Ibid.*, p. 311. The action of the splanchnic nerves on the spleen. *Ibid.*, p. 321. Some observations on the denervated spleen. *Ibid.*, 87: 189, 1936.

4. BESSEL-HAGEN, F. *Verhandl. d. deut. Gesellsch. f. Chir.*, 24: 714, 1900.
5. BIER, A., BRAUN, H. and KUMMELL, H. Bd. 3, p. 565, Operationslehre. Leipzig, 1923. Barth.
6. BLOCKER, T. G. Traumatic rupture of the spleen. *Texas State J. Med.*, 35: 478–483, 1939.
7. BONFIELD, J. P. A case of traumatic rupture of the spleen. *Canad. M. A. J.*, 36: 290, 1937.
8. BRONAUGH, W. Traumatic rupture of the spleen. *West Virginia M. J.*, 31: 363, 1935.
9. BUCHBINDER, J. H. and LIPKOFF, C. J. Splenosis: multiple peritoneal splenic implants following abdominal injury. *Surgery*, 6: 927, 1939.
10. BURKE, W. F. and MADIGAN, J. P. The roentgenologic diagnosis of rupture of the liver and spleen as visualized by thorotrast. *Radiology*, 21: 580, 1933.
11. BUTLER, E., and BIRNBAUM, W. Traumatic rupture of the spleen. *California & West Med.*, 48: 407, 1938.
12. BUXTON, J. T. Traumatic rupture of the spleen. *Am. J. Surg.*, 36: 312, 1922.
13. CONNERS, J. F. Ruptured spleens, spontaneous and subcutaneous. *Ann. Surg.*, 73: 1, 1921.
14. DACOSTA, J. C. Textbook of Modern Surgery. 10th ed. Philadelphia, 1933. W. B. Saunders Co.
15. DIGBY, L. Traumatic rupture of the spleen. *Lancet*, 2: 256, 1928.
16. DOAN, C. A., CURTIS, GEORGE M. and WISEMAN, B. K. Hemolytopoietic equilibrium and emergency splenectomy. *J. A. M. A.*, 105: 1567, 1935.
17. DODD, H. Rupture of the spleen after triflng mishaps: 2 cases. *Brit. M. J.*, 3858: 1094, 1934.
18. DRETZKA, L. Rupture of the spleen: a report of 27 cases. *Surg., Gynec. & Obs.t*, 51: 258, 1930.
19. FALTIN, R. Milzartige Bildungen im Peritoneum beobachtet ca. jahre nacheiner Milzrupture vorgenommen Splenektomie. *Deutsche Ztschr. f. Chir.*, 110: 160, 1911.
20. GARDINER, R. Delayed hemorrhage from ruptured spleen. *Brit. M. J.*, 3869: 416, 1935.
21. GORDON-TAYLOR, G. The problems of surgery in total war. *Surg., Gynec. & Obst.*, 74: 375, 1942.
22. HENDERSON, F. F. Traumatic spleens. *Boston M. & S. J.*, 183: 599, 1920.
23. HITZROB, J. M. The effects of splenectomy on normal individuals and in certain pathological conditions. *Ann. Surg.*, 69: 543, 1918.
24. HUNTER, E. A. Traumatic rupture of the spleen with delayed hemorrhage. *Brit. M. J.*, 3892: 256, 1935.
25. HOLMAN, E. Significance of temporary elevation of blood pressure following splenectomy, with particular reference to role of spleen as regulator of circulation. *Surgery*, 1: 688, 1937.
26. JOHNSON, P. Autotransfusion in traumatic rupture of the spleen. *West Virginia M. J.*, 36: 127, 1940.
27. JOLLY. Quoted by Gordon-Taylor, G. The problems of surgery in total war. *Surg., Gynec. & Obst.*, 74: 396, 1942.
28. KRAFT, R. W. Autotransfusion in ruptured spleen. *California & West. Med.*, 24: 6, 1931.
29. LEE, E. Survival of splenic tissue after splenectomy. *Lancet*, 1: 1312, 1923.
30. LOTSCH. *Deutsche Ztschr. f. Chir.*, 93: 90, 1908.
31. MACDONALD, R. Blood changes following splenectomy. *Am. J. Surg.*, 23: 514, 1934.

32. MANN, F. C. Functions of the spleen. Indiana Univ. Seminar, March, 1932.
33. MARINE, D. and MANLEY, O. T. Homeotransplantation and autotransplantation of splenic tissue in rabbits. *J. Exper. Med.*, 32: 113, 1920.
34. McINDOE, A. H. Delayed hemorrhage following traumatic rupture of the spleen. *Brit. J. Surg.*, 20: 249, 1932.
35. PENBERTHY, G. C. and COOLEY, T. B. Results of splenectomy in childhood. *Ann. Surg.*, 102: 645, 1935.
36. PERLA, D. Regeneration of autoplastic splenic transplants. *Am. J. Path.*, 12: 665, 1936.
37. PFEIFFER, D. B. and SMYTH, C. M. Late results of splenectomy for traumatic rupture of the spleen. *Ann. Surg.*, 80: 562, 1924.
38. PUESTOW, C. B. Traumatic rupture of the spleen with delayed hemorrhage. *Surg. Clin. North America*, 20: 195, 1940.
39. SHAW, A. F. B., and SHAFI, A. Traumatic autoplastic transplantation of splenic tissue in man. *J. Path. & Bact.*, 45: 215, 1937.
40. STEENROD, E. J. Traumatic rupture of spleen with delayed hemorrhage. *Am. J. Surg.*, 49: 129, 1940.
41. SUSMAN, M. P. Spontaneous rupture of the spleen. *Brit. J. Surg.*, 15: 47, 1927.
42. KUPPERMAN, VON W. Nebenmilzen nach traumatische Milzruptur. *Zentralbl. f. Chir.*, 63: 3061, 1936.
43. VON KUTTNER. *Berlin klin. Wchnschr.*, 47: 1520, 1910.
44. VON STUBENRAUCH. *Verhandl. d. deutsch. Gesselsch. f. Chir.*, 41: 213, 1912.
45. WEBB, R. C. Traumatic rupture of the normal spleen with delayed hemorrhage. *Lancet*, 59: 545, 1939.
46. WENGER, L. R. A., Traumatic rupture of the spleen. *Brit. M. J.*, 3937: 1253, 1936.
47. WOLLSTEIN, M. and KREIDEL, K. V. Blood picture after splenectomy in children. *Am. J. Dis. Child.*, 51: 765, 1936.
48. WRIGHT, L. T. and PRIGOT, A. Traumatic subcutaneous rupture of the normal spleen. *Arch. Surg.*, 39: 551, 1939.
49. ZUCKERMAN, C. and JACOBI, M. Spontaneous rupture of the normal spleen. *Arch. Surg.*, 34: 917, 1937; also RANKIN, L. M., Rupture of the spleen from muscular action. *Am. J. Surg.*, 45: 598, 1939.

DISCUSSION

W. L. ESTES, JR. (Bethlehem, Pa.): I believe the Association is to be congratulated on the delightful paper that Dr. Curtis and Dr. Roettig have prepared, and its presentation by Dr. Roettig.

We have been interested in various phases of this particular problem. Our experiences differ somewhat in the question of incidence of abdominal trauma, and also of lacerations and rupture of the spleen. We find that nonpenetrating trauma of the abdomen is encountered rather infrequently, and to many the diagnosis and treatment are not always familiar. For that reason, therefore, we believe that this is a very valuable paper, having brought to our attention various pitfalls in the possibilities of incomplete and uncertain diagnosis, and having emphasized the difference in accuracy of proper treatment and the end results. This very extensive report of Dr. Curtis is most timely on the rupture of the spleen representing a lesion that requires prompt recognition and prompt surgical intervention for a successful outcome.

Our experience has been, statistically, that of about 415 consecutive injuries admitted to our clinic. There were thirty-four of abdominal trauma, and of another group of ninety-four cases of abdominal trauma, there were seven instances of rupture of the spleen.

These cases of rupture of the spleen we have found more or less helpful to divide into three groups:

1. *Cases of severe multiple trauma*, which include rupture not only of the spleen but also of the diaphragm, liver, intestine or kidney, fracture of ribs and extremities, or lacerations of lung. These terminate fatally so quickly that very seldom is there an opportunity to apply treatment that might be effective. The trauma is so great and extensive that death is inevitable.

2. *Simple rupture of the spleen with a recognizable symptom complex*. That, again, I think Dr. Roettig dwelt upon very nicely and satisfactorily. Splenectomy is the treatment of choice, and as Dr. Roettig has shown, can be performed with a surprisingly low mortality with adequate preoperative and postoperative care.

3. *Cases with delayed symptoms or delayed rupture, with obscure or atypical manifestations*, also referred to by Dr. Roettig. These, unfortunately, are not uncommon and present a problem in diagnosis that may be very difficult to solve in time to save the patient.

Our experience with traumatic rupture of the spleen has been extremely limited. Five of these were the result of motor accidents and a fall, and one a coasting accident, and I am wondering whether that may not be a factor in the experience and incidence as between Drs. Curtis and Roettig and ourselves. I believe that in the congested, heavily populated areas, where they are many motor accidents, this injury is probably going to be found to be much more common than perhaps in the less heavily populated districts because of the high incidence of severe motor accidents.

Of these seven, three had severe multiple injuries and died within sixteen hours of the accident with no surgical treatment. Four were splenectomized. Two had uncomplicated ruptures of the spleen and survived. Two died, one as a result of concomitant contusions of the liver and intestine with terminal mesenteric thrombosis; the second had fracture of the ribs with laceration of the lung.

This in general, if any conclusion can be drawn from so few cases, corroborates Connor's dictum that mortality in rupture of the spleen depends upon the severity of the associated lesions, and that the only effective treatment is prompt splenectomy in the properly prepared patient, by transfusions, plasma, etc. It seems likely, however, with the various blood substitutes now on hand for immediate use in emergencies, that the severe complications of splenic rupture may be more successfully handled, particularly when hemorrhage is the major lethal factor.

We have also been interested in this group of cases of delayed diagnosis. Of definite interest have been various measures proposed from time to time

to aid in the diagnosis of delayed rupture. Dr. Roettig has very well stressed the point brought out by McIndoe that the x-ray has too seldom been used. With splenic rupture, in a simple flat plate of the abdomen McIndoe observed increased density in the left upper quadrant with elevation of the left diaphragm and displacement of the stomach to the right. Webb has reported similar findings.

Pendergrass has suggested that a radiograph taken after administering a Seidlitz powder will better outline the stomach and reveal the pressure deformity in the fundus that occurs with a large perisplenic clot. And finally, Burke and Madigan have likewise reported the intravenous use of thorotrast to diagnose rupture of the spleen. An abdominal film taken four hours after injection will demonstrate perisplenic extravasation of dye when splenic rupture has occurred.

We believe that the successful treatment of a traumatic rupture of the spleen would seem to depend upon the severity of concomitant lesions, the prompt combating of blood loss with equally prompt splenectomy, and early diagnosis of delayed rupture or rupture with delayed or slow hemorrhage, so that the indications for splenectomy may be recognized before it is too late.

G. Gavin Miller (Montreal, Canada): I am afraid, that I have not very much to add. There is one sign we see at times which is that of pain in the shoulder, and I think sometimes the gas bubble in the stomach with a flat plate will be shown on a straight x-ray examination. I certainly have never believed that one should do elaborate x-ray tests.

We have eight patients in our series and none of them died. They were all treated differently and they were all treated at different times. These are all cases with no multiple injuries. The patients with multiple injuries are apt to die rather promptly, and when the autopsy is done by the coroner we have no information on that series. In the cases in which we have operated upon the patients we find we have had no mortality.

I have been impressed much more by the danger of missing the diagnosis which may be a ruptured bowel. I think that early operation, splenectomy, with suitable transfusions, is a comparatively safe procedure, but I think delay in which there might be a ruptured bowel is fraught with great danger. I think that a ruptured bowel is an infinitely more dangerous condition, especially if missed, than splenectomy; so it has been our rule, where there is any doubt about the diagnosis, to explore; and I think with a suitable local anesthesia, one need not be particularly afraid of opening the abdomen in the presence of a certain amount of shock and a certain amount of hemorrhage.

Frederic W. Bancroft (New York, N. Y.): May I mention one other diagnostic point that I think is sometimes of value? We had a child brought into the hospital this year with rather an indefinite history. By flat x-ray plate, we discovered a space between the cardia and the diaphragm. We

utilized that. We gave her a little barium by mouth, put her in Trendelen-
burg position, and found a space of an inch between the cardia and the
diaphragm. I think that is a method of determining in the slow type the
presence of blood.

BYRD C. WILLIS (Rocky Mount, N. C.): I want to say a few words
about an instrument I use for collection of blood.

This is an apparatus we found very useful, and a very simple one to
arrange. In the question of collecting the blood, we found this problem: If
you use ordinary suction, you soon clot the blood and block the holes. You
have to have a special tip. We have a glass tip and a wire to protect it, and
we cover it with gauze. We have a section with the suction union at the top.
You have your citrate container on top of that, and you have your air vent
at the top.

You have at the bottom of the bottle your transfusion needle that you
run right into your patient. You suck the blood in and when you have a
sufficient quantity in, you open it up and let the blood run into the patient's
vein. We found it of very great assistance in appendix ruptures and in tears
in the mesentery, and I have recently used it (I will probably be severely
criticized) in a case of evisceration in a Negro, who had six perforations of
the bowel. I am convinced there was no leakage in the cavity because the
eviscerated bowel had the perforation, but still I used the blood and this
Negro survived.

I believe that the time is probably coming when we are going to stop
throwing away the most valuable thing the patient has, and that is his own
blood.

We have had eleven cases of ruptured spleen, and I should like to call
attention to a type of injury that I have not seen quoted before. A man
was grabbed around the abdomen by his friend, lifted up, and he did not
pay any attention to it. This was the afternoon before his admission. He was
admitted into the hospital and the internist made a diagnosis of acute
appendicitis because of pain in the lower left quadrant. But he did give one
significant symptom, that he had a pain in the left shoulder.

I operated upon the man for acute appendicitis, making a McBurney
incision. When I opened the peritoneum there was a flood of blood. If I
had not obtained the history of pain in his left shoulder, I would have been
at a loss to know where this hemorrhage was coming from. I immediately
closed the McBurney incision, made a left rectus incision, and found a
transverse rupture of the spleen.

SPRAINS AND SEPARATIONS OF THE INFERIOR TIBIOFIBULAR JOINT WITHOUT IMPORTANT FRACTURE

TOM OUTLAND, M.D.

Chief Surgeon, State Hospital for Crippled Children, Elizabethtown; Fracture and Orthopedic Chief, Polyclinic Hospital, Harrisburg

HARRISBURG, PENNSYLVANIA

WHEN the term, "sprained ankle," is used, reference is usually being made to stretching or tearing of the external lateral ligament of the ankle. In view of the rather complex ligamentous structure of the ankle and tarsus, it would be more than a little strange if all sprains in this locality were confined to the external lateral ligament. As a matter of fact, several clinical entities may be recognized by careful examination. By far the most important and most serious ligamentous injury about the ankle is sprain of the anterior inferior tibiofibular ligament, with varying degrees of lateral luxation of the ankle, but unaccompanied by an important bone injury. In the severer grades of this lesion, failure to recognize its true nature and institute proper treatment results in disability all out of proportion to the apparent seriousness of the injury. Injury to the inferior tibiofibular ligament may occur alone, but it is usually associated with sprains or tears of the internal or external lateral ligaments.

INCIDENCE

Sprain or rupture of this ligament, with consequent spreading of the mortise, is universally recognized as a fairly common complication of ankle fractures, but its occurrence without an important fracture is overlooked by most authors. Campbell[1] refers to this injury but states that it rarely occurs without accompanying fracture. Keys and Conwell[2] mention it briefly, but regard it as rare. Only two references[3,4] to this condition could be found in the current periodical literature. Older textbooks disregard it entirely.

However, Böhler[5] considers it a relatively common lesion and discusses the mechanism and treatment at considerable length. Watson-Jones states that sprains of the internal lateral ligament may be accompanied by tearing of the tibiofibular ligament. Thorndike,[6] in his monograph on athletic injuries, says that damage to the

320

tibiofibular ligament is sustained more frequently than external lateral ligament injuries. In our experience, minor degrees of this lesion have occurred about as frequently as the usual "sprained
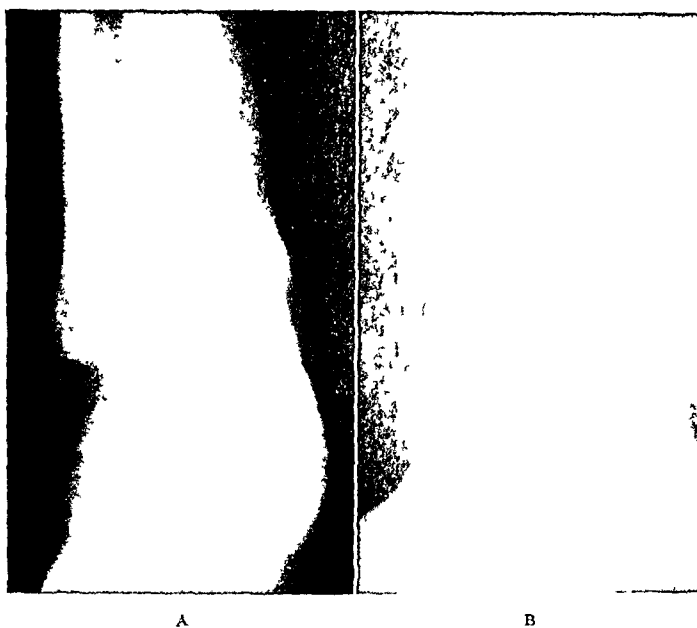
FIG. 1. A, separation of tibiofibular joint by external rotation B, roentgenogram showing displacement possible after novocainization of injured ligament.

ankle" and severe tears with lateral subluxation of the ankle are by no means infrequent. We have seen it most frequently as a result of athletic injuries—football, basketball, skiing, and the like—all having been responsible. As might be expected, it has generally affected patients in the second decade, although no age is exempt.

ANATOMY

The anatomy of the distal tibiofibular joint[7] is comparatively simple. The articulation belongs to the syndesmosis group which includes those joints in which contiguous bony surfaces are united by an interosseous membrane allowing a slightly movable articulation. This syndesmosis is formed by the rough convex surface on the medial side of the fibula and a rough concave surface on the lateral side of the tibia.

There are four ligaments in relation to this joint. The anterior ligament is a flat, triangular band of fibers which extends obliquely downward and lateralward between the adjacent margins of the

tibia and the fibula on the front aspect of the syndesmosis. The posterior ligament, smaller than the preceding, is disposed in a similar manner on the posterior surface of the joint. The inferior

A   B   C
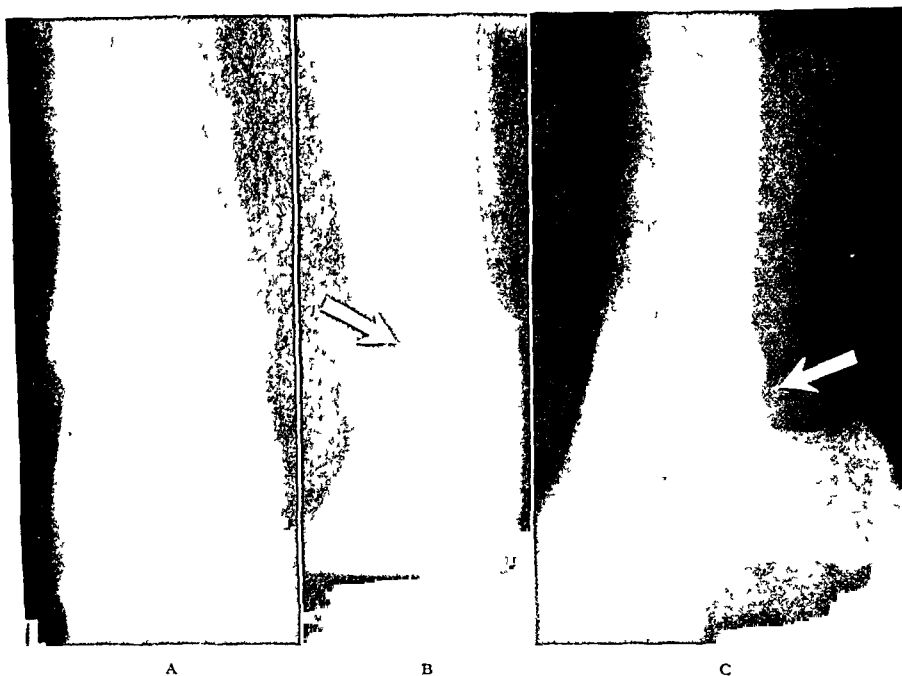
FIG. 2. A, separation of tibiofibular joint by internal rotation. B, roentgenogram after novocainization of ligament. Note separation possible. Arrow indicates small avulsion fracture. C, lateral roentgenogram showing slightly displaced fracture of posterior lip.

transverse ligament lies in front of the posterior ligament, passing transversely across the back of the joint. The fourth ligament, associated with the joint, is the interosseous ligament which is continuous with the interosseous membrane and which constitutes the chief bond of union between the bones. A small amount of motion normally occurs in the tibiofibular joint as the astragalus moves in the mortise joint during plantar and dorsal flexion. This is due to an anatomical peculiarity. The body of the astragalus is much wider anteriorly than it is posteriorly. Hence as the ankle is dorsiflexed the wider portion of the astragalus enters the mortise and a slight normal separation occurs between the tibia and fibula. Conversely, the joint narrows as plantar flexion occurs. The nature of this motion is not clearly established, but it probably consists of slight external rotation of the fibula on its long axis, and possibly a gliding motion of the fibula on the tibia. There is a normal wide variation both in the
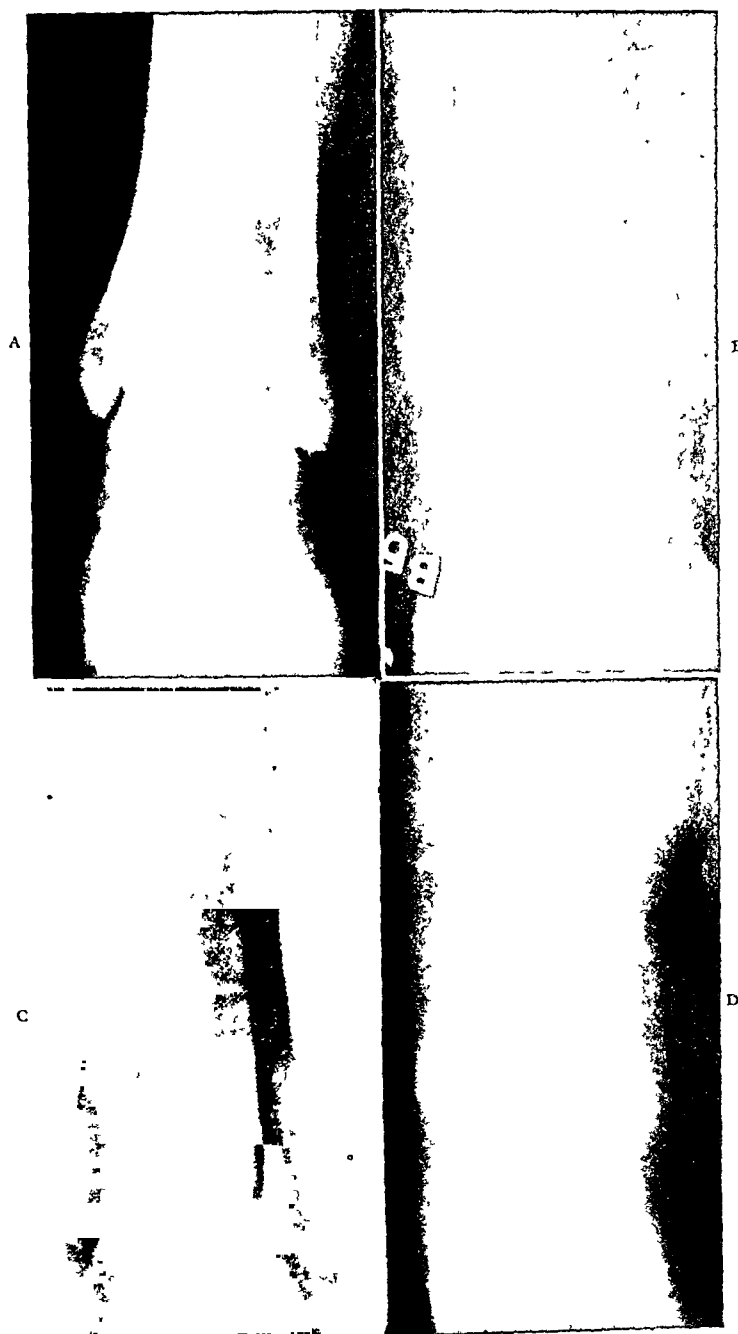
FIG. 3. A, chronic sprain of inferior tibiofibular joint. (Duration of symptoms two years.) B, roentgenogram of unaffected side. C, marked displacement possible after novocainization of ligament. D, injured ankle after autogenous bone graft, with complete relief of symptoms.

width of the joint fissure between the tibia and fibula, and also in the range of motion. It is, therefore, not possible to define the normal width of the joint or the normal amount of separation occurring during dorsiflexion, except by comparing the two sides. Neither age, sex, nor habitus seems to determine the type of joint a given individual has.

<div align="center">MECHANISM</div>

Campbell[2] states that injury to the tibiofibular ligament may be the result of either direct or indirect violence. In our experience, the lesion has always occurred as a result of rotation of the leg on the fixed foot. The rotation may be either internal or external. Böhler[5] explains the occurrence of this lesion as follows: "If external rotation occurs on the fixed foot, the astragalus acts as a lever which is rotated inward in the ankle mortise stretching or tearing the tibiofibular ligament; or the fibula may be held in place by the external lateral ligament and only the tibia rotates. In this instance, the anterior outer angle of the tibia, to which the ligament attaches, is usually avulsed. If the leg is internally rotated on the fixed foot, the ligament between the medial malleolus and the astragalus is ruptured and then the tibiofibular ligament. Often a small fragment is torn off the posterior tibial malleolus, and if the force is continued, a torsion fracture of the upper end of the fibula occurs."

<div align="center">SYMPTOMATOLOGY</div>

The symptoms naturally vary somewhat with the severity of the injury. In the less severe cases, pain in the ankle, swelling, and exquisite tenderness sharply localized over the tibiofibular joint, are found. In the more severe cases, with lateral subluxation, there is practically always inability to bear weight. While the findings on inspection are by no means as marked as in external lateral injury, the disability is much greater.

<div align="center">DIAGNOSIS</div>

In a patient with the above symptoms, whose roentgenogram is reported negative, tibiofibular ligament injury should be strongly suspected. Localized swelling and tenderness over the anatomical location of the tibiofibular joint and absence of swelling below the malleoli make the diagnosis quite certain. If the ligament is ruptured or its bony attachment avulsed, the ankle will be loose. However, due

to reflex spasm of the muscles about the ankle, it may not be possible to demonstrate laxity in the mortise joint until the pain and spasm have been overcome. This may be accomplished by the injection of 2 per cent novocain directly into the injured ligament. After a few cc. of the novocain solution have been injected, it may be possible to withdraw old blood into the syringe, showing the presence of a hematoma. As soon as pain and spasm have been overcome, abnormal lateral mobility in the ankle can be demonstrated. This is accomplished by grasping the lower leg in one hand and the heel in the other. The astragalus can now be moved laterally and medially, and felt to strike against the internal malleolus.

As before mentioned, it is wise to test the uninjured side for comparison, since the normal range of motion varies in different individuals, much as it does in the inferior radio-ulnar joint.

Radiographic Appearance. Since the displacement is a matter of millimeters, routine radiographic examinations are usually negative, although comparison with the sound side may disclose a difference in the width of the joint fissure. After anesthesia, however, the astragalus may be pushed laterally against the external malleolus, in which event not only will the joint space appear widened, also the space between the astragalus and internal malleolus may be increased.

A widened space between the astragalus and the internal malleolus is evidence of a tear of the internal lateral ligament, which is produced by abduction. If the injury to the tibiofibular ligament is by external rotation, the internal lateral ligament need not be torn; if the injury is by internal rotation, only the anterior portion (the tibionavicular ligament) of the deltoid ligament need be affected. In either event, wide separation between the internal malleolus and the astragalus will not occur, and the latter finding is, therefore, not necessary to make a diagnosis of tibiofibular diastasis. It goes without saying that in attempting to show separation, the x-ray tube must be properly centered, otherwise the oblique rays produce a misleading distortion of the joint.

In the chronic case, the findings are generally less marked, but there is a history of one severe episode with slow recovery and followed later by milder acute exacerbation due to trivial traumas. A painful snapping in the joint is often noted by the patient and may be elicited by the examiner. It is our impression that many and perhaps most of the cases of so-called "chronic sprain of the ankle" really represent unrecognized injuries to the inferior tibiofibular ligament.

TREATMENT

In those instances in which no displacement can be produced under local anesthesia and where the patient is able to walk, a zinc-gelatin boot provides sufficient compression and support. It is repeated at two-week intervals until the pain, swelling and tenderness have abated.

In cases in which subluxation can be detected, reduction and fixation in plaster of paris must be carried out to prevent the ligament healing in a relaxed condition. Campbell[2] suggests the use of a padded cast, windows being cut over the malleoli and felt pads inserted to secure a compressing effect.

In our hands, the use of a nonpadded walking cast, a la Böhler, has proved satisfactory. Under local anesthesia, as much of the swelling as possible is removed by massage and kneading. A U-splint is then smoothly applied to the lateral surface of the leg and ankle and bandaged with gauze. As setting occurs, marked pressure is exerted on the malleoli, the thenar eminences of the hands being used to distribute the pressure over a considerable area. A posterior plaster mold is then bandaged in place and the cast completed by a circular plaster bandage. Next a walking iron is incorporated in the cast and weight bearing permitted. The cast is allowed to remain from four to six weeks when it is removed and a zinc-gelatin boot applied until all tenderness and swelling have subsided.

In chronic cases, radical treatment may be necessary in order to effect a cure. Campbell[2] suggests intra-articular fusion. In our hands, the use of a bone graft from the tibia of the same side has been employed to stabilize the distal tibiofibular joint. This is driven into a drill hole in the tibia and fibula above the joint, and has proved highly successful in stabilizing the joint and in overcoming the symptoms.

SUMMARY

1. Injuries to the anterior inferior tibiofibular ligament without serious fracture are frequent, although very little is to be found bearing on this subject in the literature.

2. If a considerable degree of subluxation accompanies the sprain, disability is prolonged unless adequate treatment is given.

3. Treatment of the acute form by reduction and fixation is described; while a simple operative procedure for cure of the chronic cases is proposed.

REFERENCES

1. LEWIS, DEAN. Practice of Surgery. Vol. 3, chap. 3, p. 30. Hagerstown, Md., 1937. W. F. Prior.
2. KEY, J. A. and CONWELL, H. E. The Management of Fractures, Dislocations and Sprains. St. Louis, 1934. C. V. Mosby, Co.
3. KIRSCHENMANN, J. J. Rotated Fibula. N. Y. State J. Med., 37: 1731, 1937.
4. ALLDREDGE, RUFUS H. Diastasis of the distal tibiofibular joint and associated lesions. J. A. M. A., 115: 2136–2140, 1940.
5. BÖHLER, LORENZ. The Treatment of Fractures. P. 455. Baltimore, 1936. Wm. Wood & Co.
6. THORNDIKE, AUGUSTUS, JR. Athletic Injuries. P. 60. Philadelphia, 1938. Lea and Febiger.
7. GRAY, HENRY. Anatomy of the Human Body. Pp. 341–343. Philadelphia, 1936. Lea and Febiger.

DISCUSSION

LLOYD NOLAND (Fairfield, Ala.): I am afraid I have not very much to add. The subject has been covered very well indeed, and I think that Dr. Outland has given us a most interesting paper.

Diastasis of the distal tibiofibular joint must, of necessity, be associated with extensive ligamentous rupture and may occur without associated major fracture. The mechanics, I think, are open to question. Whether or not this can occur with internal rotation, I question, but most of us are inclined to associate such an occurrence with Potts' fracture in which there is fracture of the internal malleolus or rupture of the internal lateral ligament with fracture of the lower third of the fibula. We must all agree with Dr. Outland that failure to recognize and properly treat the diastasis may, and often does, result in permanent disability.

Aldredge states in a paper read before the American Medical Association about two years ago, that definite widening of at least 2 to 3 mm. takes place at the tibiofibular joint when the foot is fully dorsiflexed, and that when the tibiofibular ligaments are sectioned, the fibula separates abnormally from the tibia when on dorsiflexion the foot reaches 90 degrees. It, therefore, seems evident that long immobilization in slight plantar flexion is indicated in every case in which separation with ligament injury is suspected in that joint. Just how long one should immobilize these patients, I do not think anyone knows. You have to try them out, I suppose. If you set an arbitrary six weeks of immobilization when there are rather violent symptoms, perhaps that is all right. There are patients, of course, on whom you will have to have metallic fixation later, but I do very firmly think the matter of putting on these braces in slight plantar flexion has a great deal to do with the end result and certainly tends toward comfort of the patient.

ROBERT L. RHODES (Augusta, Ga.): I had the pleasure of reading Dr. Outland's paper, a copy of which he sent me, and as though that were not

full enough, we have his pictures and his movies which just leave very little to say.

I do have a few things that I want to suggest, mainly and particularly a slight modification in the treatment from that which he has suggested.

Dr. Outland is to be congratulated for bringing so clearly and so thoroughly to our attention a lesion often overlooked and associated with persistent pain over a prolonged period of time. However, I wish to emphasize his sentence, "The exquisite tenderness sharply localized over the tibiofibular joint" as the key in diagnosis.

For many years I have treated these badly sprained ankles, even some associated with milder type fractures, with lateral moulded lightly padded plaster splints instead of the U splint. The external or lateral splint is applied first, and extends from high up on the leg under the foot to the inner side of the instep. In other words, the lateral splint comes down and under the foot, whatever position you put it in, leaving a gap in here on the medial splint rather than carrying the U all the way around. This mesial splint is held snugly and carefully moulded until it "sets." The internal or mesial splint is then applied from a corresponding position on the leg down to about one-half or one-fourth inch above the other splint, depending upon the amount of swelling. This also is held snugly and carefully moulded until it "sets." A muslin bandage or sometimes adhesive straps in addition, is firmly wrapped around the splints. I have not had quite the success in moulding out as much of the swelling as others have, so I have used sort of a delayed measure. The muslin bandage is then replaced from day to day as the swelling recedes and the gap in the two splints allows those to be brought closer together.

In about a week or ten days all swelling is gone, the joint is snugly in position and circular plaster is applied instead of the muslin bandage. Using the moulded splints as a whole for compression, tightening them from day to day, has avoided the necessity of the windows and pads as suggested by Campbell. After their removal an elastic anklet or elastic wrap bandage is worn for several weeks.

WILLIAM DARRACH (New York, N. Y.): I think this is a very important lesion. We have been of the opinion for a long while that it was an important lesion, whether you have malleolar or high fibular fractures with it or not, and I am delighted to hear Dr. Marble say he had not seen one and did not know it existed, because I did not think there was anything in this line he did not know about. They are not common, I will grant that, but there are plenty of them and he has seen them but he has forgotten.

Two or three points on the treatment: I believe that four weeks or six weeks is entirely too short, because I think it takes a long while for this ligament to heal and get solid, and I think they have to be off weight-bearing, with any stress and strain on it for a longer period than that. Whether it should be eight weeks or twelve weeks, I do not know. That is the conservative treatment.

That is why for a number of years, because of this long duration, we have been cutting off a little time by putting a bolt through the two malleoli, holding the thing firmly together. At first in that soft cancellous bone, we found the head on the one end of the bolt and the nut on the other end began to cut into the bone, and we started putting plates on the end. Using those, we can start the ankle motions earlier and get them up on a walking brace at the end of three or four weeks. The bolts are usually taken out after six or eight months.

RALPH G. CAROTHERS (Cincinnati, O.): I think this is terribly important because there is a lot of talk going around that all you have to do is put in a little novocaine and tell them to go on out dancing that night; and doctors are doing that sort of thing every once in a while, then some of the rest of us get this situation when it is in a bad chronic state.

If an ankle is truly sprained in the true sense of the word, just a little stretching of some ligament without a true rupture or a true breaking of bone structures, I suppose that a little dab of novocaine, or perhaps a little colored medicine on the outside would do; but when things are really ruptured inside, as Dr. Outland has pointed out, this greater care is required. Or it may be a lesion such as I worked on last winter, that is, a rupture of the astragalofibular ligaments, which gives you almost the same thing, inasmuch as when they are ruptured and you put a little novocaine in and twist the foot in inversion, you can rock that astragalus over until it is off line about 25 or 30 degrees. Either one of those lesions requires a plaster cast and a long continued treatment, and not this proposition that the coaches are trying to put over, that is, just a strap and get the fellow back in the game the next day and perhaps have him out forever.

THE BURN PROBLEM*

Lieut. Col. I. S. Ravdin, m.c.†

Diplomate of the American Board of Surgery

PHILADELPHIA, PENNSYLVANIA

THERE is even now no universal agreement regarding the treatment of minor, much less, the extensive superficial burns in spite of the fact that these lesions are as old as fire itself. Not only is there no universal agreement, but there is no agreement in this or any other country. Every surgeon is constantly confronted with new agents, or old ones in new skins, purporting to overcome the deficiencies of those substances already available. Careful, unbiased investigations can, and should be carried out to arrive at conclusions which clinicians can use to guide them in the selection of methods and agents for the treatment of the burned area.

The lack of agreement in regard to the local treatment of the burned area is similar to lack of agreement on the treatment of wounds in general. The American Red Cross Manual gives instructions that a weak tincture of iodine be used in a wound, a procedure which must result in further tissue injury. There can be little doubt that the extent of injury in a burn is further increased by certain methods of therapy.

There is, however, agreement on many of the major factors which are involved in the pathologic physiology of the burned patient and these may be used as a basis for outlining the general therapy.

It is agreed that the pain which the extensively burned patient has is an important factor in the induction, progress and the prolongation of the shock to which these patients are subjected. The burned patient should receive adequate doses of morphine. An adequate dose is one that will relieve pain and it should be remembered that the burned patient tolerates relatively large doses of morphine.

The burned patient rapidly loses a part of his plasma volume. A portion of this is lost to the exterior, but the largest part is lost into the tissues in and surrounding the burned area. It is possible that small additional amounts of plasma are lost in other portions of the

body, perhaps through the mediation of some toxic agent absorbed from the burned area but this is relatively unimportant in the early period of therapy. It is the loss of plasma volume into and around the injured tissues which is the principal cause of shock. Shock can, in large part, be prevented by the administration of plasma, serum, human albumin or some other adequate blood substitute. Even if shock has occurred it may be overcome by restoring a depleted blood volume with substances which tend to remain in the circulation, provided the shock has not existed so long as to make the state irreversible. It is best, therefore, to initiate the use of blood substitutes early, before shock in the traditional sense has become manifest. The cause for irreversibility in the shock reaction is even now not clearly understood. To wait for a low blood pressure and a failing circulation before initiating therapy designed to maintain a nearly normal plasma volume is to court disaster.

The use of blood during this early period may be harmful, for plasma continues to be lost while the red cells remain in the circulation. This leads to homoconcentration and an increased viscosity of the blood, to a deficient oxygen carrier, greater work on the heart, and thus to an intensification of the shock. While whole blood is useful when anemia occurs it should *not* be used in the first few days of treatment unless a *severe anemia is present. In burns associated* with other wounds transfusion may be necessary at any period of therapy.

It is now generally agreed that the administration of large amounts of sodium chloride in hypoproteinemia intensifies the edema which occurs at any given level of serum protein. The patient with an extensive superficial burn rapidly becomes acutely hypoproteinemic if he receives intravenously large amounts of sodium chloride, because a further loss of plasma protein from the vascular system occurs as the sodium chloride leaves the vessels, carrying with it a variable fraction of the plasma protein. It is because of this that it has been suggested by the Burn Conference of the National Research Council that the patients receive no more sodium chloride intravenously than they have received plasma, or a comparable blood substitute, in the same twenty-four hours. Local therapy is valueless unless sufficient plasma, serum, albumin, or a comparable substitute is utilized to maintain a plasma volume sufficient to permit the continuance of a nearly normal cardiovascular function, a normal, or nearly normal fluid exchange between the tissue fluids and the blood, and normal respiratory and cerebral functions.

The local treatment of the burned area is the most debated of all aspects of the "burn problem." Every conceivable form of local application has been recommended and applied. Complete sight has been lost of the undeniable fact that a burn is a wound, and that the fundamental principles governing wound treatment should, wherever possible, be used in the local treatment of the area of the burn.

While initially the area may be sterile, it is readily susceptible to every type of contamination to which wounds in general are exposed. It is for this reason that these wounds so frequently become infected. They are not protected adequately immediately following the injury, and they are too frequently not protected adequately during the emergency and definitive treatment. This factor must be continually stressed in the training of house officers.

The débridement of the badly devitalized tissues must be done with the greatest care lest living tissue be further injured, and with the healthiest respect for asepsis. Here indeed is an area which provides a perfect culture medium. The specific local therapy should be designed to discourage bacterial growth and to prevent further contamination. I am sure that extensive débridement under local anesthesia is rarely necessary. It is far better to leave behind small areas of nonviable epidermis and dermis than to remove viable areas from which regeneration must take place. The extensive scrubbing procedures without doubt cause more shock than the good which they do. The removal of loose skin and the gentle washing of the surface with gauze, good white soap and plenty of sterile salt solution is about all that is required. The addition of one of the sulfonamides to the material which is to be applied locally should serve further to reduce the incidence of suppuration.

Since a portion of the plasma which is lost from the circulation is lost from the surface of the burn, the local therapy should be designed to minimize this. The local tanning of the area provides a satisfactory method for attaining this objective. It is equally important, however, that the tanning agent does not simultaneously injure remaining islets of epithelium from which repair can take place. Not only must these residual islands not be injured, but repair must not be retarded by the locally applied agent. There can be little doubt but that tannic acid and gentian violet or the other dyes do, to some extent, inhibit cellular repair and perhaps cause some further necrosis. Whether they actually cause the extensive necrosis of otherwise viable tissue which has been ascribed to them cannot now definitely be answered, but I do not believe that they cause this necrosis except

perhaps when used with relatively high concentrations of silver nitrate.

In spite of the objections it should be recognized that the tanned area, as a rule, requires minimal care and permits of rapid evacuation, considerations of great importance in military surgery. If one considers further that patients so treated do not require as large amounts of plasma as comparable cases which are not tanned, it becomes necessary carefully to weigh the advantages of tanning against its disadvantages.

The pressure dressing method, which has within recent years gained a number of advocates, has a great deal to commend it. With this method an ointment containing a bacteriostatic agent, such as one of the sulfonamides, can be placed on the burned area following débridement and the part firmly supported and bound. Plasma loss is decreased by this method, the patients without doubt remain comfortable, subsequent tissue injury is minimized, and subsequent dressings kept at a minimum.

Whether this method will prove to be useful to our armed forces as it is now proving to be in the care of burns in civil life remains to be determined. It has much to commend it, for it treats the burned area as a wound. We shall have to keep our minds open in regard to this method of therapy. Surgeons in civilian hospitals should adopt it for certain patients so that we may learn further of its advantages and disadvantages. For the face, hands and genitalia it will, without doubt, be helpful, for the use of tanning solutions on these areas has led to much needless deformity. It is time that we generally agree to condemn this practice.

While the local and perhaps oral sulfonamide therapy will reduce the incidence of infection, these substances cannot be expected to correct gross errors in surgical care. If an adequate amount of a potent sulfonamide is used locally, I doubt the necessity of oral therapy. The burned patient frequently has a low renal output and great care should be exercised when those substances are used which tend to crystallize out in the urinary tract. Failure to heed this warning will lead to anuria from mechanical ostruction.

When infection occurs the local area must be treated as one would treat any other infected wound, by wide open drainage, thorough irrigation, and the local and oral use of bacteriostatic agents. To permit infection to gain firm foothold is to jeopardize the life of the patient, to prolong convalescence or to increase the resulting deformity.

It is important that the area of a third degree burn be skin grafted as soon as possible. This will reduce the incidence of infection, for such areas invariably become infected if not grafted. It will minimize deformity and will provide a healthier area than is obtained when epithelial proliferation from a few residual islands and from the wound edges is depended upon to cover the area.

The modern development of technics for obtaining grafts of varying thickness and extent for covering such areas should do away with the unsightly "pinch grafts" which are even now too frequently used for covering the denuded area. Such plastic procedures require some additional knowledge, but the surgeon who does not possess this should not attempt the reconstructive stage of therapy.

The intelligent care of the burned patient requires an intimate knowledge of basic surgical principles and a working knowledge of surgical physiology. While the intern may possess the latter, he does not have the former. These patients are often relegated to the youngest house officer for care when in reality they deserve the attention of the most experienced member of the staff.

It is highly likely that burns will constitute a considerable group of the casualities in our armed forces as well as in civilian life during the next few years. We must meet the problems which they present without bias and prejudice.

<center>DISCUSSION</center>

WILLIAM DARRACH (New York, N. Y.): I hope I never have to be in the position of living on the gleanings of a field that Ravdin has harvested, because he leaves very little for the gleaner.

There are one or two things that I would like to emphasize, however, and that is, I think with this problem of burns, the thing which he brought out, and on which I would like to place the greatest emphasis, is that burns are wounds and should be treated on the same fundamental principles that wounds are treated.

Secondly, I think that we can divide our present day knowledge into the things we know and the things we do not know. Of the things we know, I think first comes what he said about pain. Pain must be relieved because it makes the patient sicker if it is not, and not only pain at the time but pain during the ensuing days and weeks.

The second thing I think has been pretty well cleared up and that is the question of the loss of the blood constituents. Most of these patients do not need red cells, most of these patients do not need salt; and if we think of what we heard this morning about the autopsies on the badly burned patients, the kidneys and the kidney tubules, we will not give them too

much salt. But what they do need is plasma, and plenty of plasma. I think that is pretty clear and definite.

I think another thing on which we all agree is that one of our most important duties is to prevent infection, and to prevent infection by means which will do good and not do harm, that is, by means which will get rid of the contamination with as little damage to tissue as possible.

What he said about gentle washing with a soap solution instead of violent scrubbing is along the same lines. He spoke of the use of strong chemicals to get rid of bugs which at the same time kill body cells. I think we are all in agreement on that.

There is another fundamental principle in the treatment of wounds that we all agree on: they all need rest, and that means general rest and local rest.

I think one of the things we are not sure about is what to do with the dead tissue. We realize that in compound fractures and in contaminated wounds, the object is to get rid of the devitalized tissue and the foreign material. How far shall we apply that in burns? I think we do not know to what extent we should débride and get rid of the dead tissue. That is why the tannic acid treatment which seems clinically to have helped so tremendously in the ways in which it has been used, perhaps is theoretically wrong. I have not been able to think that thing through; I do not know. Here we are covering this wound, or continuing the covering of the wound by tissue which is practically dead. Perhaps that is wise; perhaps it is not.

On the question of coverage, I think clinically everybody agrees that we must get these wounds covered in some way to cut down the plasma loss, to cut down the pain, and to hasten healing and quiet down the edema. What is the ideal coverage? I cannot help but believe that theoretically, at least, the ideal coverage is skin. Now if means can be worked out whereby skin grafts shall be done at the earliest possible moment instead of later on, I think we will make advances in this treatment. It has been done in some places but I do not think we know yet how early it can be done safely.

As for the sulfonamide drugs, I want to put in one word about those and I like to say it as often as I can in regard to their use in wounds, whether compound fractures or burns or anything else: Let us remember that they are aids and not substitutes for good surgery.

JOHN F. GILE (Hanover, N. Y.): There is very little that I can add to this discussion after Dr. Farmer's paper, Dr. Ravdin's paper and Dr. Darrach's discussion. About the only thing I can do is to agree with everything that has been said. I do not think there is any question about everybody's agreeing with the first-aid treatment, getting these people warm, and the use of morphine and plasma, and so forth.

As far as what you are going to put on locally, I think there is still, of course, considerable discussion. We believe that in patients who look as though they were going to have considerable dehydration, tannic acid is the

best treatment. On the other hand, as has been emphasized here, we believe that it should not be used on the extremities, and so forth. We have people coming in, upon whom the treatment has been decided on the outside by the first-aiders with a very generous use of tannic acid. We think that perhaps somewhere in the Red Cross book, that might be changed somewhat. We have had cases come in, in which we wouldn't primarily pick tannic acid as the treatment, on fingers and so on, on which it has been put, and we are then pretty well sold as to what the method of treatment must be.

We believe that steariform and wet saline solution with pressure is about as good as anything we have; and we are of the opinion that patients get along and look about as well as with any other type of treatment. That is what we are using with pressure dressing, particularly, and splinting at times. We have also recently in two or three cases on the lower extremities—and I see no reason why it couldn't be used on the upper extremities—put on the splints, so you get the legs up; and where there are bad burns on the back of the leg, you get it off the bed and up so you can dress it, and it is certainly more comfortable.

We have used no sulfa drugs in oil. We have used the powder on some of these burns and believe that it cleans them fairly well. But I think one other word of warning should be given. You should not continue to use it too long, as we believe that after a certain time it inhibits new growth. Whether that is absolutely true or not, I do not know, but we have rather believed so. We agree absolutely that early skin grafting is very important, getting them grafted as soon as possible, and starting motion, and so forth, of the part as soon as possible.

DOUGLAS ACKMAN (Montreal, Canada): I should like to describe the method we are using at the General Hospital in Montreal. Before going into details, I might give you a little preview of how it occurred. Because of the emergency conditions and the fact that we are surrounded by munitions plants in Montreal, and the remote possibility of air raids, it was considered necessary to organize some kind of plan. In doing this, we had a committee which operated, and we have achieved a certain working plan which, with the help of a few slides, I would like to show you now.

We have one chart which gives the timing in the treatment. The timing object presupposes the development of the treatment as the burn itself progresses. We have to have two teams operating at the same time, one a shock team on a twenty-four-hour basis, and the other a burn team for local treatment, both under the supervision of a senior surgeon.

This chart also outlines laboratory examinations and clinical data. We have divided our burn timing into a period of shock—the first forty-eight hours; the period of toxemia—the second to the fifth day; infection and granulation between the fifth and fourteenth day; and finally, a healing time which we put from the second to the sixth week. Our object is, first, to

treat the patient's shock before attempting to undertake local treatment, and the treatment is shown on this chart.

Just one word about the treatment with plasma. It is controlled by repeated hemoglobin estimations. We have to have a hemoglobin estimation done every hour for the first four hours, and thereafter every four hours, especially in severe cases. In the second hour, if the case is desperate, we give cortin, 25 cc. intravenously in extreme cases, and continue that cortin 10 cc. intramuscularly as indicated. Plasma is given according to a plan.

In the local treatment which is divided into two parts, one sees the employment of an eschar treatment for trunk, thigh, legs, feet, arms and forearms, and on all critical areas we are using a sulfathiazole emulsion which has been developed at the General Hospital.

The redressings that are done are also shown on this chart. In the second twenty-four hours, it is sometimes necessary to redress our critical areas, and at other times it is not. It depends upon the serum loss and the appearance of the dressings. During the second twenty-four hours we have a complete blood chemistry done. In the second period of toxemia, the sulfonamides are introduced if they are needed, blood substitutes as indicated, and if in the eschar we have infection, it is unroofed and the emulsion dressing applied. All critical areas continue to be redressed at suitable periods.

In the period of infection or granulation, as the case may be, we carry out the treatment according to plan. If anemia develops, transfusions are used, and diet and vitamins as indicated. Again if infection occurs, it is unroofed and the emulsion dressing applied.

We have believed it is better, no matter what type of eschar is used, that it be removed if necessary by surgical measures at the end of the second week, and thereafter if it is a second degree burn, it is treated by emulsion dressings. If it is a third degree burn and requires skin grafting, this is done after the use of saline dressings until the granulations are suitable.

A second chart, which we are using, is to control the treatment. The nurses in attendance on the ward have these charts in front of them; they do them, themselves, and they have the basic chart to go by. One column indicates blood pressure, and another the pulse reading. Hemoglobin estimations are carried out in these cases. There are one-hour periods, and consequently everything is done on a systematic basis. For notations regarding treatment, and for purposes of laboratory examination, we use other indications on the chart. On the back of the chart an estimate of the burns is given.

The emulsion which we have been using at Montreal General Hospital is an oil and water emulsion with some microcrystals. The technic we are employing is as follows: We clean the wound under anesthesia, preferably, using morphine only, and lavage it with saline solution, spray on some powder as a frosting, and then use the emulsion in single thickness strips of

gauze, either ordinary hospital gauze, or better, a curtain mesh, which we call a sulfa mesh. These can be made beforehand or made up as you dress. We apply the dressings over these and use some padding. We have had no experience with pressure dressings but we do use plaster moulds.

CHARLES LUND (Boston, Mass.): I first want to thank the society for the invitation that I have had to attend the meetings, and to say how much I have enjoyed them. I do not want to prolong this discussion too long, but I have had a recent experience that I think is of some interest. In trying to treat the class of burns that is now essentially insurable, we run into new problems when we use these new methods. Two weeks ago I was confronted by a man burned by flaming alcohol which got onto his cotton shirt and burned him completely front and back, from the waistline of his trousers up.

The face and the hair were only first degree. About a tenth of that area, on the chest, was a third degree burn and the rest was a bad second degree burn, and there was also a circular second degree burn of the right thigh, and a small patch on the left thigh. We calculated about 70 per cent of the body was burned. The genitalia were not burned and the legs below the knees were not burned.

Recently, a patient died in Boston where the medical examiner believed that compression of the chest from a circular eschar was a major contributing part of the cause of death. I knew about that case and because I was impressed by some things I learned at a meeting in Cincinnati, I tried to use boric ointment with pressure. You cannot put on much pressure when you are dressing with boric ointment completely around the chest.

The wound was cleaned, shock was treated during the cleaning, and then enormous dressings were put on. There was tremendous leakage of plasma for the first two days, and during that time, 2,000 cc., twenty units of 25 per cent albumin were given, diluted in 10,000 cc. of saline. That brought his hemoglobin, which was 128 two and a half hours after injury, down to 100. Following this, he was given saline, his hemoglobin sank to 80, and his urine output went way up to 3,000 and 4,000 cc. a day. He was given sulfadiazine by mouth and he remained in what looked like good condition.

On the fifth morning he began to breath a little heavily and I did not like his looks. I thought he might be getting pneumonia. There was no way to examine him for pneumonia on account of the burn. I consulted with an excellent pneumonia man, and we did not do anything very active. Later that day a plasma protein was taken for determination, and at 9 P.M. he was dead. He died with definite respiratory signs the last two or three hours. The plasma protein came back the next day, 3.2 albumin and 1 per cent globulin, a total of 4.2 in a laboratory where the normal is around 7.

That man should either have had blood plasma or whole blood during the last forty-eight hours of his life, and we thought because his urine output stayed up so well, he looked so well and his hemoglobin was so high, that he did not need it.

I think we will find that this additional test is important and must be repeated frequently and reported promptly. This perfectly splendid chart covering everything might possibly suggest that you do a little more work and get plasma proteins in.

I think that is about the biggest dose of human albumin that has been given. That dose, incidentally, is the product of forty pints of blood.

FREDERIC W. BANCROFT (New York, N. Y.): I have some cases* showing a fatality which I think are worth while reviewing now:

A child with a burn of two days' duration came into the Lincoln Hospital and was débrided. In the upper end there was one little area of epithelium. There was edema in the subcutaneous tissues and down to the large vessels in the back. This shows very definitely, I think, the amount of edema that occurs in the subcutaneous tissues in the vicinity of a burn, and where we could save a lot of our plasma loss and prevent edema from occurring.

Another case is that of a six-day burn in which infection occurred rapidly. At the upper end, where the burn was, nothing but a parchment membrane, a little dark line, appeared. That was the separation between the living and the dead tissue. This must be débrided and treated.

We have also seen one of the rare cases of ideal treatment of the eschar, showing the eschar up above, the correct location of the sweat glands no infection beneath the membrane and a firm splint of the membrane. I think that not only is an eschar of advantage, but if we débride that eschar, we must handle the tissues gently in order to preserve the layer of epithelium.

GROVER C. PENBERTHY (Detroit, Mich.): I do not want to prolong this discussion, but the treatment of burns is a timely subject and Dr. Ravdin has emphasized the importance of the surgical care. Dr. Darrach reëmphasized it, as have others, so I would like to speak for a moment and refer to the work that Dr. Johnston is doing in Detroit for the National Research Council.

This morning we discussed the frostbite of the mariner. Now we may have the same problem to deal with in the case of the mariner burned with oil. A very thick engine oil had been applied to the burn. That burn with oil is treated with ordinary lard and an emulsion formed, and after that emulsion has been completed, the wound is thoroughly washed with soap and water and irrigated with saline or boric acid. This prepares that wound for the coagulant, whether you use gentian violet or tannic acid.

Dr. Darrach said he wondered what should be done in the way of débridement. I think it should be the necrotic treatment, the blisters opened, and so forth.

Dr. Kreuscher asked the question whether the emulsion was applied as a dressing and I can answer no, just a cleaner. The President referred to the Pearl Harbor situation. There they had many oil burns to deal with.

* Dr. Bancroft presented illustrations of these cases.

E. Payne Palmer (Phoenix, Ariz.): The first problem in the severely burned patient is the treatment of shock. Many of these patients can be saved from sudden death and moved to the hospital by the standard shock and burn treatment—these fifty-seven varieties we have of burn treatments —if they are treated for shock at the site of the accident and not moved until the condition is improved. Too many of these patients are moved while they are still in the state of severe shock, and the mere fact of turning them or attempting to move them may terminate life right there, or the transportation to the hospital may terminate life.

In these various demonstrations here, you give them morphine, which is all right; you give them the cortical extract; but you must give something that is going to get the patient out of shock as quickly as possible. As I said this morning, and I am going back to it again, give big doses of coramine to get the patient out of that immediate shock, and when you get him out of that period, you can give the coramine intramuscularly in order to get him into the hospital. It is only a temporary affair, but you must have something which you can give these patients until they get into the hospital to have the standard treatment which you are going to give them there.

But do not move these patients in their state of shock until their condition will permit them to get into the hospital, until their muscle tone is improved, until their venous tone has improved, and until the flow of blood in the veins has improved.

Hubley R. Owen (Philadelphia, Pa.): I will not prolong the discussion any longer except to say that from 1937 to 1940 on our service in Philadelphia General Hospital we had the opportunity of treating firemen for extensive burns. We tried all methods and the method we used more extensively than any other was the one mentioned by Dr. Wagner, the ambrine method or paraffin, and tannating these wounds. But I think the important thing we have gathered from this is to treat the patient first and the burn after.

I cannot come around to using engine oil on these burns. It does not sound like good surgery, any more than using Carron oil, because it must be dirty, you have a primary wound to deal with and you will get infection if you use Carron oil or engine oil. I do not like the sound of that.

THE TRANSPORTATION AND OPEN
TREATMENT OF SEVERE FRACTURES

JOHN R. NILSSON, M.D.

Professor of Surgery of Trauma, College of Medicine, University of Nebraska

OMAHA, NEBRASKA

IN the first World War, we learned much about the transportation of patients with severe fractures. During the first two years, all kinds of splints were used: metallic, wood, and plaster of paris. Many times severe infections, hidden abscesses under plaster dressings, atrophied muscles, and gas gangrene developed which necessitated amputation.

The proper first aid and transportation of a patient with a fracture has a marked influence on the end result. Love's statistics of the World War show that more than 30 per cent of all war casualties admitted to the evacuation hospitals were compound fractures. The British, during the first two years of the war, had a mortality of 50 per cent in compound fractures of the femur. They used principally the Liston wood splint. After discarding this type of splint and adopting the Thomas traction splint, their mortality rate dropped to 15 per cent.

The Allied Medical Staff adopted the Thomas, Murray-Jones, and Kellar-Blake splints. They were put into service and proved very practical and efficient. They were made of iron and were light, unbreakable, and hundreds of them could be placed on a truck and be delivered to our Field hospitals. Their application to an extremity was very simple. It is said that every soldier in the British Army was taught how to apply a Thomas splint before he was sent into the firing line. Our Army and Navy are using these same splints in the present war.

ADVANTAGES OF TRACTION AND SUSPENSION IN TRANSPORTATION

The advantages of traction and suspension in transportation are: (1) The lack of pain; (2) ease in handling and transportation; (3) accessibility to wounds for treatment; and (4) no damage to muscles, nerves, or blood vessels during transportation.

From personal experience during the War and also since that time, I am convinced that these are the best first-aid and transportation splints available at the present time.

341

Every case of fractured extremity that came to our hospital during the last year of the World War had a Thomas, Murray-Jones, or a Kellar-Blake splint applied, and in many instances the treatment was continued in the original splint.

A   B

FIG. 1. Mr. A. O., age fifty-four, slipped on the ice; comminuted fracture of the right humerus; two gold Parham bands applied. The patient made an uneventful recovery. Bands still in place fourteen years later.

On my service at the American Red Cross Military Hospital No. 3 in Paris, 72 per cent of all cases were compound infected fractures, and all of them were treated by débridement, Dakanization, traction and suspension in one of the above splints. In the present war, sulfonamide therapy will be used: sulfanilamide in the wound and sulfathiazole by mouth to combat infection. The first experience of this treatment on a large scale by our American surgeons was at Pearl Harbor where all compound fractures were treated by débridement, sulfanilamide in the wound, sulfathiazole by mouth, and the extremity placed in a Thomas or Murray-Jones traction splint. All the surgical teams were impressed by the results of the sulfonamide therapy. Drug reactions were rare. No tetanus developed as tetanus toxide had been given on entrance to service. Several cases of gas gangrene developed in which wounds had been closed by suture (which was contrary to Army instructions). These patients were treated by débridement, sulfonamide therapy, and x-ray. No amputations were necessary. Dr. Moorhead, who was in charge of one of the surgical teams, says, "The splendid results obtained at

Pearl Harbor depended on (1) Early treatment within 6 hours; (2) team work; (3) available Plasma for shock treatment; (4) débridement—no suturing of wounds; (5) sulfonamide therapy."

A   B   C
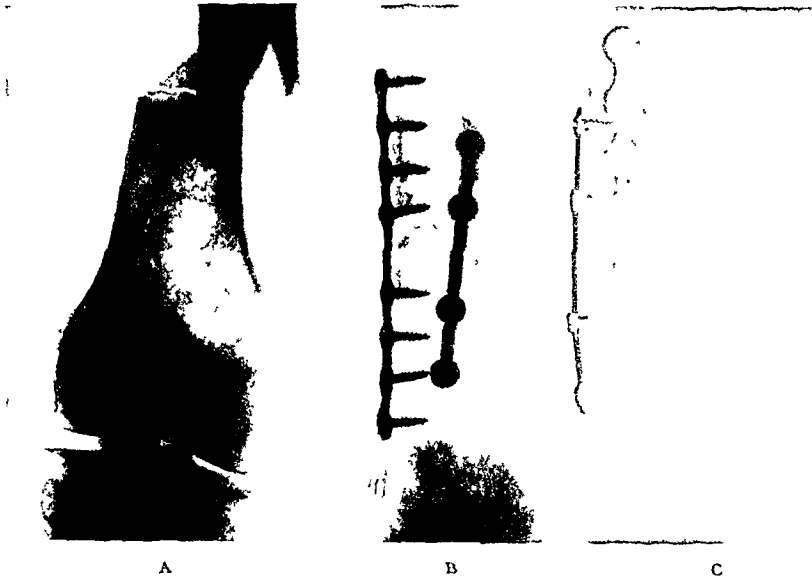
FIG. 2. Fracture at the junction of the middle and lower third of the femur. Tongs were applied, then two Lane plates.

A   B
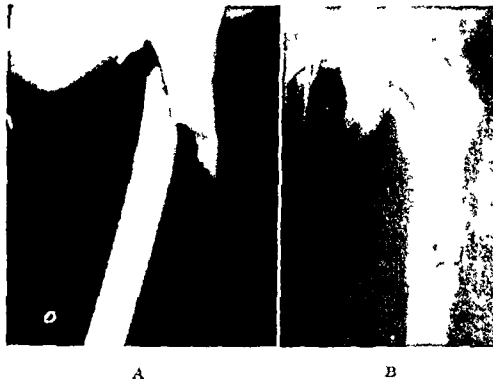
FIG. 3. A, fracture of the upper third of the femur of a child four and one-half years old. Parham band applied; B, four years later.

The open reduction of severe fractures is now more generally used than the closed method. This is evident to anyone attending the larger clinics. The reasons for the increase are: (1) We are treating more severe fractures than we did twenty-five years ago. (2) Every severe fracture is a potential liability to the surgeon in charge. (3)

With the sulfonamide therapy, i.e., sulfanilamide in the wound and sulfathiazole by mouth to combat infection together with the improved instruments, plates, screws, and technic our results are better.

A   B

FIG. 4. E. B. B., age thirty-five years, fell a distance of forty feet from a bridge on May 16, 1926; causing an oblique fracture of the upper third of left femur. Open reduction—Parham band applied. leg in Hodgin's splint; uneventful recovery. Band still in place sixteen years later.

It is interesting to review some of the older textbooks on the treatment of fractures which bring out this gradual change from the closed to the open method. Scudder's Sixth Edition, published in 1907, mentions only three cases treated by the open method; one a fracture of the olecranon, another a fractured patella, and the third a fracture of the surgical neck of the humerus. A silver wire was used in all of these cases and no mention or illustration of a Parham band or a Lane plate is found in this edition. In his Eleventh Edition there is page after page on open reductions and illustrations of the Parham band, plates of various kinds, screws, pins, etc.

My first ten years of practice were spent in a mining district. At that time we treated all of our patients with severe fractures by the closed method, using either molded plaster splints or plaster casts.

During the World War we treated our patients with severe fractures in traction splints. Shortly after my return from war service,

FIG. 5. J. K., age twenty-five, fell from the top of a box car on October 20, 1936, causing comminuted fracture of the distal end of the tibia and fibula of the right leg with forward dislocation of tibial fragment on astragalus and a linear fracture distal end of left tibia with no displacement. Open reduction right leg using Lane plate and aluminum bronze wire. Discharged from hospital December 23, 1936. Plate and wire were removed.

I became associated with Dr. A. F. Jonas, then Chief Surgeon of the Union Pacific Railroad, who taught that a good anatomical result was necessary in order to obtain a good functional result. We all
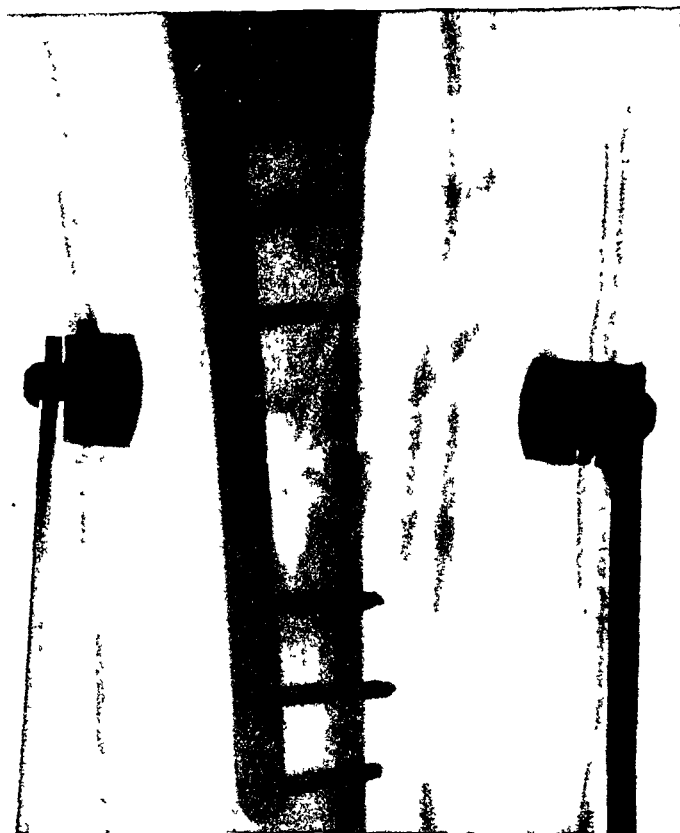
FIG. 6. Mr. O. K., age fifty-nine, July 26, 1942, compound comminuted fractures of tibia and fibula of right leg, junction of upper and middle third Open reduction July 28. Stainless steel plate with screws through both cortices (as advocated by Dr. Clay Murray). A Thomas splint with traction was applied. August 15, the wound was healed, and the leg was placed in a plaster cast extending from toes to mid-thigh, with incorporated walking iron. He was dismissed from the hospital August 16, walking with crutches

know that the two main objectives in treating any fracture are to prevent deformity and to restore normal function. Dr. Jonas did many open reductions using Parham bands, Lane and Sherman plates, and aluminum bronze wire.

A few years ago the British Medical Association appointed a committee to investigate relative results in fractures, open and

closed methods. Their conclusions were as follows: (1) Nonoperative in children 90.5 per cent good functional results, operative in children 93.6 per cent; (2) progressive depreciation of functional results from nonoperative as age advances; (3) operative treatments give higher percentage of good results at all ages; (4) the best way to get a good functional result is to secure a good anatomic result; (5) a method that does not definitely promise a good anatomic result should not be chosen; (6) for best results operate early, not after failure of non-operative treatment; (7) operative treatment requires skill and expense; (8) the mortality rate is practically nil.

In industrial work such as I was engaged in, it is absolutely essential that our reductions are as nearly perfect anatomically as possible; because if there is any liability in connection with an injury and an unscrupulous lawyer would show an x-ray film in which the fragments of a fracture were not in perfect alignment, it would make a most damaging Exhibit A against us.

At the January, 1941, meeting of the Committee on Fractures and Other Trauma of the American College of Surgeons at New Orleans, the open reduction of fractures was discussed by Dr. Murray, of New York, Dr. Venable, of San Antonio, Dr. Berg, of Pittsburgh, Dr. Morrison, of Boston, and Dr. Speed, of Chicago. They expressed their views in regard to the most satisfactory metallic plates and screws to be used in these open reductions. Dr. Venable reported the results of his research work showing the influence of the tissue fluids on the various metallic plates used at the present time, which were as follows: (1) The microammeter shows that Vanadium steel produces 200 milliamperes; (2) stainless steel produce 50 to 100 milliamperes; (3) stainless steel passified by HNO_3 produces 10 to 20 milliamperes; and (4) vitallium produces 1 to 2 milliamperes momentarily then 0.

The amount of electrolytic disintegration of metals and consequent erosion of bone seem to be related to the current flow recorded by the microammeter. A metal that will produce many milliamperes will produce much erosion of bone and the metal itself will disintegrate. A positive proof of electrolytic action in the body is the ability to recover constituent metals of an alloy from the tissues adjacent to it.

Dr. T. A. Carnes, of the Republic Steel Corporation, has shown by x-rays that vitallium plates and screws, which are made of cast metal, contain air bubbles and are more easily broken because of these defects. The Republic Steel Corporation in their research

laboratories are working on different combinations of elements for plates which promise to be more ideal for fracture work than any plates on the market at the present time. All metallic plates, now available, show some electrolytic action between the plates and the body fluids, some to a greater extent than others.

The Sherman Vanadium steel plates, according to some authorities, corrode; sinuses form and the plates have to be removed. I understand, however, that molybdenum has been added to the metallic constituents of the Sherman plates and a new stainless steel plate is the result which is subject to very little, if any, corrosion by the body fluids.

During the past twenty-five years, I have used all kinds of metallic plates, screws, Parham gold bands, and silver and aluminum bronze wire in my fracture work, and the only cases in which I have had to remove the plates were in those in which sinuses had formed. These sinuses I attributed to low grade infection, loose screws, or improper fixation. Since the report made by Dr. Venable on the electrolytic action of the body fluids on the plates and screws, I believe that perhaps the occasional necessity of removal of a plate may have been due to this cause. Very few removals, however, have shown any evidence of corrosion.

Dr. Berg in his twenty-nine years of experience with the Sherman plates, states that he believes it is the technic of application of the plate rather than the kind of plate used.

In conclusion, I will state that in severe fractures we will have fewer permanent deformities, better functional results, and the period of disability will be shorter when an open reduction has been performed. The success of the internal fixation of fractures will depend upon: (1) The careful preoperative preparation of the patient, i.e., the field to be operated upon should be cleaned and tincture of merthiolate applied twenty-four to forty-eight hours before the operation. (2) Preoperative preparation of splints, open plaster casts, molded plaster splints, or splints such as Thomas, Kellar-Blake, or Murray-Jones should be available where traction or suspension is to be used. (3) Spinal anesthesia is the anesthetic of choice in fractures of the lower extremities. (4) Rigid fixation is secured by the use of a chromium nickel steel plate with screws extending through both cortices. (5) The end result will depend upon the experience, intelligence, and good judgment of the surgeon in charge.

TREATMENT OF MINOR CASUALTIES

WILLIAM DARRACH, M.D.

Diplomate of the American Board of Surgery; Professor of Clinical Surgery, College of
Physicians and Surgeons, Columbia University

NEW YORK, NEW YORK

MANY interesting laboratory studies and clinical experiments are being carried out as to the best method of treatment of burns, compound fractures and shock. The reports from Spain, England, Germany and Pearl Harbor show that distinct advances have been made since the last war in the handling of major casualties. To those of us who remember our sad efforts to combat shock in the last war it is a great joy to know that there are already available to the armed forces 150,000 packages of dried plasma with the means of administering it under the crudest conditions. This number before long will reach 600,000. The effects of the sulfa drugs on the outcome of abdominal wounds and compound fractures is another source of encouragement.

We hear little, however, about the handling of a group of casualties which for two reasons should receive more attention. I refer to the so-called "lightly wounded" or "minor casualties." They deserve more thought because they not only comprise the majority of all injuries but they are the only ones who have much chance of getting back into service. The figures of the last war show that 53 per cent of the casualties were soft part wounds of the extremities without fracture or injury to the large vessels or nerves. Added to these were many similar wounds of the head, chest, back and buttocks. From the standpoint of numbers they deserve primary consideration.

Of all the penetrating wound of the head, chest, abdomen, spinal cord and of the compound fractures, the number who ever returned to active duty is very, very small. If one of the main duties of the Medical Corps is to maintain the strength of the fighting forces, it seems rather silly to focus attention on the group who are permanently out of the picture.

Though we may differ on some details of wound treatment, we will all agree that the time between the trauma and the operative treatment is one of the most important factors in determining the period of disability and the successful result. This holds true of

the minor wounds as well as the major ones. Yet we have all seen long queues of lightly wounded, waiting and then passed farther and farther back while the operating rooms were filled with patients with serious wounds of the chests, abdomens and compound fractures and a valiant but usually unsuccessful attempt made to restore them to duty.

We all accept the wisdom of débridement for compound fractures and penetrating wounds of the thorax and abdomen and yet forget that it originally was proved of value on minor wounds. If a small superficial wound be débrided early and sutured, the disability period may be measured in days. If it is neglected until infection is well established, the victim may be incapacitated for months; yet these patients rarely get anything but a dab of iodine and a change of dressing during their long period of travel.

If the problem were faced in cold blood and courageously, the methods would be exactly reversed. These lightly wounded should have the right of way and the time and attention of the best surgeons at the first operating station they reached. After they had been attended to, the serious cases should have their chance. However, the numerical strength of the fighting forces is not the only responsibility of the Medical Corps. Morale must also be maintained. If the soldiers learned that the seriously wounded were being put to one side, if the people at home learned that their boys were being neglected, there would be trouble brewing. My plea is not that such a complete reversal be made, but that plans should be made in each advanced hospital for the prompt surgical care of both types.

The handling of the minor casualties differs in several ways from that of the major cases. In the first place they rarely need preliminary shock treatment. Secondly, the operative procedure is much simpler. In both groups the stay in the operating room may be divided into three periods: (1) the preparation, (2) the operation itself and (3) the application of dressings, including splints. In the major cases, the second or operative period occupies a majority of the total time, perhaps 70 per cent. In the minor cases this middle or operative period may be no longer than the other two. Experience in the last war seemed to show that operating teams handling the major cases would do well if they averaged one case an hour. With proper organization it was demonstrated in several places that one operating surgeon could handle six to eight minor cases an hour. In most operating hospitals the usual practice was to have one table for each team and all three stages were done by the operator and his assist-

ants. Sometimes, by using two tables, with an extra Corps man, the first and third periods of preparation and splintage could be made to overlap the actual operating. In a few places by assigning extra assistants and three tables to one operator, the minor cases were put through at a much more rapid rate. If intravenous anesthesia were used, it would be possible to have the preliminary work done by a well trained Corps man or nurse. The same holds true of the third period or application of dressings. That is, the Ford assembly line principle is applied to this group. If inhalation anesthesia is necessary, a second anesthetist would be required.

The after treatment of the minor cases requires far less nursing and a considerable proportion of them would become ambulatory within a few hours or days. This would mean adapting ward service and arrangements to the different groups. Different plans for the convalescent period should be made according to the conditions under which the total hospitalization scheme is working. My understanding of the present Army plan is to have a surgical hospital assigned as far forward as possible for the so-called "non-transportable" cases. This would fulfill at least some of the humanitarian duties of the Medical Corps. The problem of restoring men to active duty will fall largely on the shoulders of the evacuation hospitals. It is hoped that in these units plans will be made to divert the stream into two separate channels to be handled as described above. Under certain conditions it might be wise to have a separate hospital assigned to each type.

This principle of giving the lightly wounded an even break can be applied in our home defense plans as well as to the problems of the armed forces. No one can foretell how much of what has happened in England will be repeated here, but plans to meet catastrophes are being made in most of the larger centers. Arrangements have been made in most hospitals for meeting a sudden demand for handling considerable numbers of injured people. These injuries may be from bombs, explosions, train wrecks or other causes. It is hoped, as plans to meet these emergencies are made, it will be provided that these lightly wounded will receive the same prompt, early operative treatment as the more serious cases. Most of the minor casualties can be handled on stretchers and do not need the more elaborate set-up of the formal operating room. It would seem wise to have separate teams assigned for the two types of work.

In meeting this problem either at the front or at home one of the most important considerations is the proper primary sorting of cases.

One of the most experienced men on the surgical staff should be stationed at the receiving center in order that he may decide to which group each case should be sent. The lightly wounded could be sent directly to the operating group appointed for this purpose. The more severely wounded, divided into three groups, would be sent either (1) directly to the shock wards, (2) directly to the major operating room, or (3) be made comfortable during their brief period of stay.

SUMMARY

Much attention is being paid to the handling of burns, compound fractures and shock and the use of the sulfa drugs for war casualties. We hear little, however, as to the treatment of the lightly wounded which not only comprise the majority of traumatic conditions in wartime but are the best possibilities for early, complete return to duty.

The time between injury and operative treatment of wounds largely determines the period of disability. One duty of the Medical Corps is to maintain the numerical strength of the fighting forces. Although very few of the seriously wounded ever get back to duty, they not only have received priority in treatment but have occupied most of the attention of surgical teams. Minor casualties should get at least an even break. With proper operating room organization, six to eight minor cases an hour can be handled as compared to one major case an hour. After-care and convalescence can be provided on a much simpler scale than for the seriously wounded. Plans to handle minor injuries should be made in hospitals at home if any catastrophe causes a sudden rush of injured people.

DISCUSSION

H. GURTH PRETTY (Montreal, Canada): I hardly think that Dr. Darrach's paper requires discussion. The author has pretty well completed the arguments pro and con. It is interesting and important and to the point.

One important feature is that he refers not only to the armed forces, with regard to the lightly wounded, but also to the civilian or munition forces in connection with the lightly wounded, and I think that point should be stressed. Most of us are inclined to let some first-aid person or nurse segregate the cases coming to the hospital, and the result is that the moderately lightly wounded or slightly injured munitions' workers are relegated to the out-patient department where a junior house officer does the work, with the result that at some later time they are admitted

to the ward with some form of infection, cellulitis or otherwise, which considerably incapacitates them and keeps them from returning to duty.

The other point that Dr. Darrach stresses is very important, and that is the question of two front-line surgical centers, one for the severely wounded and one for the lightly wounded, which is equally important, and again stressing the importance of the supervision of a trained surgeon for the segregation and operation upon these patients.

I can well recall the latter station. I was a medical sergeant and anything less severe than a compound fracture seemed to be left entirely in my hands. I had no medical knowledge and I dispensed as I saw fit, and I certainly realized that many of the men must have been incapacitated by my over or underindulgence in treatment.

Just a word on the lightly wounded from the point of view of more or less civilian and munition accidents. There is one point which I would like to stress, and that is, in the last three months, I have run into three more or less lightly wounded individuals. One was an Air Force mechanic who, on coming home for the week-end, cut the flexor longus to his thumb, not with a beer bottle but with a ginger ale bottle. This made me particularly disgusted because we are not allowed to touch any of the armed forces, although they are in Canada, and they are supposed to report to the Air Force personnel. This happened at midnight. He was not able to get in touch with any of his Air Force personnel and he finally came to me at the hospital where we repaired the tendon, kept him for a day or so, and the Air Force immediately took him in hand and reprimanded me for attempting to suture the flexor longus tendon.

The next was a munition's worker who works in a shell-filling plant, putting fulminated mercury in percussion caps. On one occasion some of these caps exploded and he was literally peppered with little particles of brass turnings and filings and bits of bronze. He was treated lightly at the plant, merely by dressings, only to find out that about three weeks later he was suffering from copper poisoning.

The third was a ballistics engineer who learned ballistics from his own point of view. During the trial of a Maxim gun, somebody pointed the gun in the wrong direction. The bullet went through two oak partitions of the office and finally struck this unfortunate individual just above the antero-superior spine on the right side and tore into the loin making an area about three to four inches in diameter. The interesting thing about that is that he learned ballistics from his own point of view, and the other thing was that they were keen enough then to see that immediate surgical procedure was carried out and they did not attempt anything further.

I think the point made here by Dr. Darrach is the strict surgical appreciation of these minor wounds, both in the armed forces and the civilian worker.

RUPTURE OF THE DIAPHRAGM DURING A POWER DIVE*

DESCRIPTION OF A NEW METHOD OF REPAIR AND CASE REPORT

FRASER B. GURD, M.D., C.M.

MONTREAL, CANADA

W E have all heard of the period of "blackout" suffered by members of the crew during power or dive bombing and rumors, at least, of the methods adopted for its control have been current. Recently, I have had under my care an aviator who presented a lesion due to power diving which, to my knowledge, is new. If rupture of the diaphragm occur at all frequently, and particularly if the pilot of the machine be the victim, a possible added explanation of crashes under these circumstances may have been discovered.

On August 12, 1941, I first saw A. K., thirty-nine years of age, who had a few days before come to Canada as an Air Force member of the Czechoslovak Military Mission. His history showed that prior to 1938 he had been a member of the Czechoslovak Civil Air Force and had been examined and re-examined medically while in this service. In 1938, he joined the Military Air Force and was again re-examined. Following the invasion of Poland he escaped from Czechoslovakia and after incredible hardships was successful in reaching England. He was again re-examined before becoming active in the Czechoslovak Squadron attached to the R.A.F.

He took part in the Battle of Britain and in mid October, 1940, while a gunner on a plane over Brussels and while taking part in a power dive, he suffered a sudden onset of pain in the chest, nausea, shortness of breath, and a shock reaction as indicated by cold perspiration and a sensation of extreme weakness. He apparently did not lose consciousness. He was admitted to a Naval hospital in the south of England where a diagnosis of ruptured diaphragm was made. The urgency of his symptoms subsided and he was informed that, although operative interference was required, the surgeons in England, because of air-raid conditions, were unwilling to undertake fancy surgery.

He remained inactive until about mid summer, 1941, when he flew to Canada in a bomber. During the trip he again suffered from acute symptoms of pressure in the thorax and upon his arrival in Montreal was referred to me for treatment.

He looked ill, and stated that he had lost about twenty pounds in weight in the interval since October, 1940, that he had attacks of faintness and suffered from a loss of appetite with nausea but apparently no vomiting.
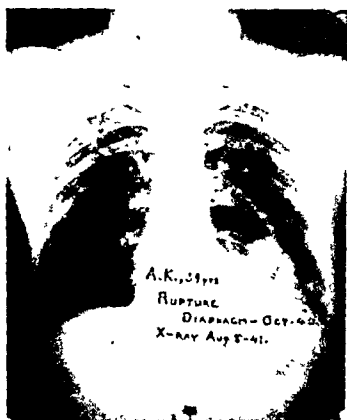
FIG. 1. Flat plate shows hernial sac above the heart shadow.

FIG. 2. Barium filled stomach. The organ lies high in the thorax and demonstrates a typical "upside-down" appearance.

He carried with him a flat plate of the thorax which showed the presence of a hernia occupying the greater part of the left chest reaching well above the arch of the aorta. Examination of the chest showed parts to be tympanitic, other parts to be dull; there was an absence of breath sounds over the left side of the chest except for a smallish area behind and above. (Fig. 1.)

He was admitted to The Montreal General Hospital where further x-ray examination, following barium administration, proved the stomach to be located well above the arch of the aorta and proved, also, that the splenic flexure was close to the top of the chest. (Figs. 2 and 3.)

Operation was recommended and eagerly accepted. It was carried out August 17th. Thoracotomy was accomplished through the bed of the seventh rib which was removed from the transverse process to its anterior end. The sixth and eighth ribs were cut so that adequate exposure within the chest was easily obtained. Under the posterior part of the opening a thin layer of completely atelectatic lung was discovered. Under the anterior two-thirds or more of the incision I came directly into an enormous sac. In the cavity, which gave the impression of being about the size of the operator's head, was situated the whole of the stomach, practically the whole of the transverse colon to which was attached the great omentum, the splenic flexure and much of the descending colon. Numerous coils of small intestine were present as were the spleen and the left lobe of the liver.

All of these structures were easily pushed down into the abdomen through the very large hiatus except the stomach which was adherent to the upper and posterior part of the sac over an area about the size of the

FIG. 3. Barium filled splenic flexure is shown lying close to level of the clavicle.
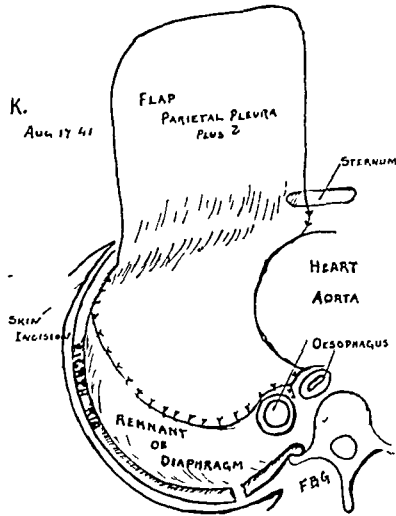
FIG. 4. Diagram drawn at operation illustrates the size of the opening in diaphragm and the method adopted to fill the defect.

operator's hand. I must admit that while engaged in the somewhat trying problem of separating the stomach from the upper part of the sac I felt rather embarrassed since it did not seem possible that I should be able to close the enormous opening between the abdomen and the thorax.

Once the abdominal viscera had been replaced in the abdomen I was left with what I estimated to be about one-quarter of the diaphragm in position in the posterolateral corner and with an opening roughly circular in shape about 20 to 25 cm. in diameter.

If it were not possible to close this opening, the patient's life would have been jeopardized without profit. I decided, therefore, to separate the parietal pleura from the endofascial layer over the anterior aspect of the chest. Incisions were carried vertically up through the parietal pleura to close to the dome of the thorax and joined by a transverse incision. The flap thus outlined was comparatively easily separated from the underlying fascia and periosteum and was found to have considerably more stability than I had expected. It was evidently composed, I believe, not only of parietal pleura but, in part at least, of peritoneum and in all probability of shreds of diaphragm. This adequate flap was turned down and sutured into position to the remnants of the diaphragm and somewhat carefully about
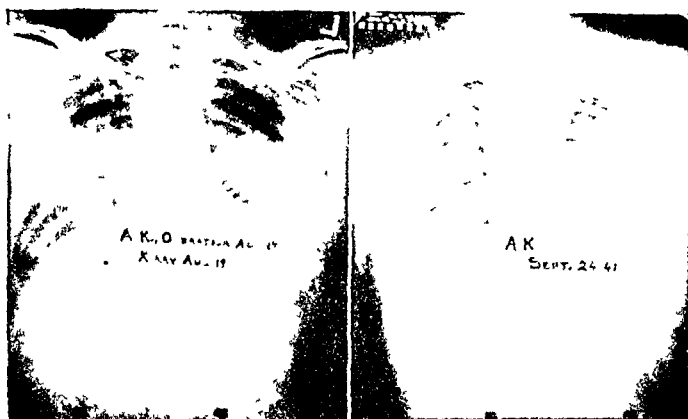
FIG. 5. X-ray film forty-
eight hours after oper-
ation. Absence of any
pathological lesion on the
left side and atelectasis of
right middle lobe is clearly
seen.

FIG. 6. X-ray film five weeks
postoperatively.
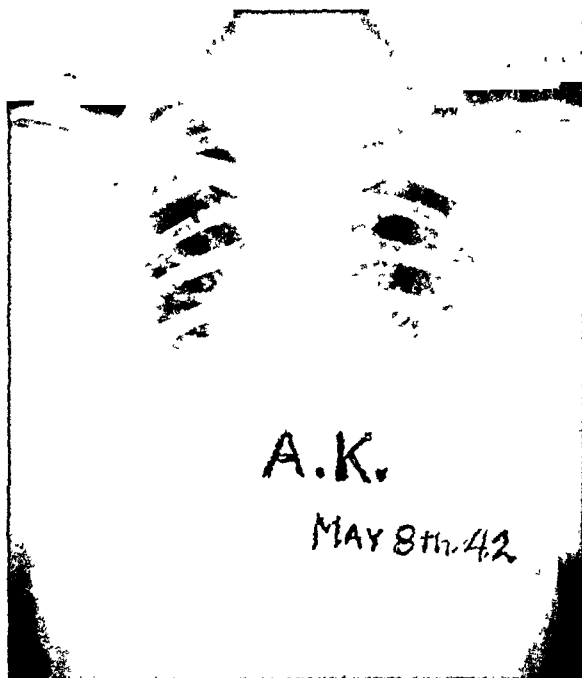
FIG. 7. X-ray film eight months postoperatively.

the esophagus and aorta. For this purpose interrupted black silk sutures were employed.

At the completion of the operation the inside of the chest looked ship-shape although the lung showed no tendency to come down and fill the

FIG. 8. Photographs of patient eight months after operation.

cavity. (Fig. 4.) Although absolutely no movement of the slip of diaphragm present was exhibited, the phrenic nerve was crushed as it lay on the pericardium.

The wound of the chest wall was closed with interrupted fine black silk sutures with silkworm gut sutures and clips to the skin. Four heavy catgut sutures were employed to approximate the sixth and eighth ribs.

Following operation the patient's condition remained very satisfactory although he developed a localized area of atelectasis, probably in the middle lobe, on the contralateral side. (Fig. 5.) He was kept in an oxygen tent for two days and thereafter rapidly improved in his general condition. Aspiration of the left side of the chest on the second day resulted in the recovery of 400 cc. of slightly blood-stained fluid. He was out of bed on his tenth day and discharged from the hospital on September 11th. (Fig. 6.)

He was examined May 8, 1942. At this time he had put on forty-nine pounds weight since operation. He exhibits no digestive disturbance whatever and does not suffer from shortness of breath upon exertion. He returned to duty with the Czechoslovak Air Force Training Center in Canada on October 15, 1941. His only complaint is that he has been "grounded." Although I have advised against battle aviation, I am certain that he will demand that he be permitted to fly with such insistence that his superior officers will give in. Examination of the chest showed absolutely no lag on the operated side and normal breath sounds are heard throughout. X-ray examination of the chest showed no abnormality. (Fig. 7.) There is no disfigurement of the torso and no disability. (Fig. 8.)

SUMMARY

A case is reported of a middle-aged aviator who during a power dive ruptured the left diaphragm. Complete destruction of at least three-fourths of the organ occurred. Massive herniation of abdominal contents into the thorax took place.

Reconstruction of the missing diaphragm was accomplished by a method which I believe to be original. For this purpose an enormous pedicled flap consisting of parietal pleura was employed.

DISCUSSION

WILLIAM J. BRICKLEY (Boston, Mass.): I am privileged to be invited to discuss this case. This was a difficult job well done, and with a good result.

I am a medical examiner. I have been for twenty-two years. My patients give me no history, and mostly my work is by deduction. They cannot tell me, so that I have to deduce a great deal. However, I have been a surgeon. I have examined 90,000 of the living and I have only taken care of 10,000 of the dead so far. I do autopsies and I deal with the big hospitals. I love surgery, I love medicine, and I talk with the fellows that do it. Dr. Gurd's was a good job. That is the first thing I would say.

Next, I am going to discuss the surgery of it. I am going to tell you a little bit about hernia, its diaphragmatic formation. I have talked many times to my own group about it. Hernia is produced, as I see it, by a weakness and then a force. Almost never do I see a hernia produced by a force in an uninjured or a congenitally strong belly wall. I recall the case of a man who jumped off our Customs House, came down 400 feet, landed on concrete; all his bones were broken. He was broken the way you throw an orange against the wall. He had no hernia; his liver was ruptured; all his bones were fractured.

I remember an aviator who came down 500 feet and hit the ground. They do have diaphragm ruptures, and have them very frequently, and I see them I would say two or three in every hundred autopsies that I do. They are not unusual.

The place that the diaphragm ruptures is the left side, either anterior or posterior, and there is a mechanical reason for it. I see the diaphragm so often it is before me as I think of it. At eleven o'clock I was poking my finger through the esophageal opening of a man who had died, so that I would know what I was talking about.

The diaphragm is higher on the left side than on the right side. The left side comes up to the sixth rib, and the other side, the right, comes down to the fourth. The diaphragm on the right side is a little heavier than the diaphragm on the left side. The diaphragm on the left side has a larger plaque. It is a central tandem. You remember there is a right and a left and a central, that is the clover-leaf type. The crust on the left side is not

as good as the crust on the right side. The crust on the left comes down to the sixth lumbar. The crust on the right comes down to the first, second and third lumbar.

There are three holes in the diaphragm. I am speaking mechanically and perhaps I can speak figuratively. The one on the right is the vena cava. The vena cava of the ordinary person is about the size of a broomstick. It would be about an inch or an inch and a quarter in diameter. When there is internal pressure, it is exerted by the liver. The liver is soft and squashes up against the diaphragm and tends to shut off the inferior vena cava. You know when they operate upon the gallbladder, they pull it up, and I wondered why they did. I know. It is because the upper part of the inferior vena cava goes through a deep groove.

On the left side we have the esophageal opening which is near the spine. The level that is shown in a diagram is a higher level than is shown in the diaphragm. Quite often that is so, because the esophagus comes down the center as far as the fourth, and then it begins to go off toward the left, and along about the eighth to the tenth, it begins to leave and go forward and to the left go through a hole in the diaphragm.

The lower end of the esophagus has a sphincter and it is a sort of cuff-like sphincter. There are three of them, top, middle and lower, but the lower not only shuts it this way but it puckers it up against the hole in the diaphragm so that it forms a plug, just the way we would repair a hernia and we would pull something in to plug it. Within the chest you have a negative pressure, within the abdomen you have a positive pressure, so if the hole through the diaphragm at the esophageal opening is large enough, you will tend to come out, first by suction from above and next by pressure from below.

The esophageal opening would admit my finger, but it has to be big enough to have a bolus go through it. Of course, it will always vary, but it goes through at a double angle, through a curved surface and from front to back, going outward and downward. Now it goes through the muscular part in the back of the diaphragm, near the central raphe, but that central raphe on the left is fortified by the pericardium, and hence it is hard to tear that. It is a good central raphe so that you will not get a tear at or around the pericardium unless a person is shot. You will not get it by stresses or strains because it is heavy, but you will get a tear of the muscles on the left side, the muscular part of the diaphragm along the left border, either where it is attached or at its curvature.

The liver is one of the most variable organs we have in shape, size and contour. Sometimes the right lobe of the liver would point up steeply. It would be like the top of a derby hat, and the left lobe of the liver would be like a brim; it would be an inch or two, only, and you may or may not have a left lateral ligament. You have five: right, left, central, coronary and falciform to hold it in place. The right ligament is always good, and the coronary is always good. The left one, however, may be absent. The left

lobe of the liver would be as thin, sometimes, as my finger, and the other part of the liver would be a dome that would run up high, the whole liver being completely within the right chest.

Further, in order that the esophagus may pass down through the diaphragm, the left lobe has to be anterior. On the right it will not come down. That forms a weak place so that with any intra-abdominal pressure the epicardium on the right is safeguarded; on the left, no. It is out of the way. It is either small or is in advance of the hole.

The esophagus itself has a cuff-like lower sphincter and that varies very, very greatly, and the stomach is not as the pictures are drawn. That is, you have not a big cardia with a large end in it. That only occurs after death or when you have a lot of gas. In many stomachs that we see, the esophagus comes down and there is only a little cone until it is distended. I can always drag the end of the stomach or the part of the stomach adjacent to the esophagus into the chest cavity. You do it easily. That hole is like a button-hole, and it is so that when the diphragm pulls, it is shut off. You cannot swallow and breathe at the same time. That is the reason, because your diaphragm shuts it and it will not go through when you pull it down wrong, but if you pull it by inadvertence or accident or otherwise, the esophagus gets above the diaphragm—that is if there is slack enough to do it—and you are in trouble for the esophagus itself is thicker, much thicker than the adjacent part of the stomach, though the esophagus spreads out in the wall of the stomach in a trumpet shape and it is thickened there. If you pull it past that thickening, it stays there, so that often, in doing an autopsy, we find the cone of the stomach has come up into the thoracic cavity, maybe an inch or two or three inches.

Now given that condition, intra-abdominal pressure will tend to make it worse. The peritoneum does not stretch much but it stretches a little. It will not stretch as big as your head. When you are sewing up a fat wall of belly and you are pulling the peritoneum over and you take a stitch, it will tear out if you put too much tension on it. In the hernias we see, they are formed with the peritoneum; they are formed little by little with the gradual stretching, and then, as many of us know, you can have a stretch as big as your head. It will hold an ovarian tumor. We see it in the ventral tumors as big as your head. Therefore, my deduction in the matter is that there is first a weak place, and then a forcing out. It is little by little on top of a small defect.

What are you going to do about it? I would suggest to those who see aviators with that condition, which has been determined either by x-ray or otherwise, that they think twice about it. They should compress his abdomen to see whether, under such compression, he might go up; for when he comes down or decelerates, as they call it, that is, comes to the low part of his flight and increases his abdominal pressure, something is likely to project above the diaphragm. You will never project it there because you

have an adequate negative pressure, but you always have a positive pressure pushing it up, so I would suggest that it might be wise to have such a man examined.

I know that I find many of them. About 2 or 3 per cent of those that I see have some sort of cone-shaped projection, and I am only speaking from 2,800 autopsies, so there is something to it.

The manner of it, I will not take up; that is, the force necessary to develop it, but as a mechanical proposition the left side of your diaphragm is not as good as your right side, and in case of pain on the left after an event of this sort, I think it would be wise to think of hernia of the diaphragm.

FRASER B. GURD (closing): I might just say that you will all have recognized that I resisted the temptation to have diagrams drawn with arrows pointing in different directions to show how this rupture took place.

ANESTHESIA FOR THE SURGERY OF TRAUMA

SIDNEY CUSHING WIGGIN, M.D.

Diplomate of the American Board of Anesthesiology; Chief Anesthetist, Boston City
Hospital; Director of Anesthesia, The Faulkner Hospital; Instructor in Anesthesia,
Harvard Medical School

BOSTON, MASSACHUSETTS

HAD we discussed anesthesia for the surgery of trauma five years ago, we would have considered the increasing number of traffic, industrial and home accidents. Today, however, we must in addition to these, concern ourselves with the casualties associated with the world wide conflict in which our country is now involved. In this regard, we find our problems quite different from those of the first world war in that civilians as well as our armed forces are subject to attack.

We are confronted with a problem quite different from the last war, namely, that of civilian casualties, besides the wounded soldiers. Developments in anesthesia during the past ten years have contributed much to the improved care of civilian casualties in peace time. First the physician anesthetist has trained himself, not only in the improved anesthesia technic, but in the evaluation and treatment of the patient's general condition and in this way is able to relieve the surgeon of much responsibility. For instance, of great importance is the consideration of shock in these patients who have been subjected to loss of blood, trauma to the nervous system and tissue damage. This is treated with plasma, venoclysis, or transfusion before any anesthesia or surgery is considered. The tissues of the patient, who has recently been in shock, have already suffered impairment of function from anoxia. It is highly important that the anesthetist keep this in mind and furnish the highest possible concentration of oxygen during the operation. Preliminary preparation alleviates the fear of the anesthetic and operation, thus reducing the metabolic processes so that the anesthetic when given is made more efficient and safe. The drugs used for premedication are morphia and atropine for ether, pentobarbital sodium with atropine or scopolamine, for the gas mixtures and spinal. This reduces the incidence of difficult induction, irregular course of anesthesia and has provided better relaxation and postoperative recovery.

Today we have a wide choice of anesthetic agents and their combinations, while not many years ago we were limited to ether for routine use. After much trial and error the choice of drugs and methods has become well established. This has been brought about by the recognition that efficient anesthesia demands a balancing of drugs, anesthetic agents and oxygen. Carbon dioxide absorption, brought forward by Waters in 1926, has revolutionized the administration of gases. In 1929, spinal anesthesia was re-established on a more scientific basis; as a result we may now secure relaxation with safety in the most difficult abdominal procedures and operations upon the lower extremities. In 1930, avertin was introduced as a rectal anesthetic. Since 1934, the rapid acting intravenous barbiturates, evipal sodium and pentothal sodium have been developed as general anesthetics for short operations. Since the re-advent of spinal anesthesia physician anesthetists have utilized combinations of all these various anesthetics with oxygen.

Regional and intratracheal anesthesia have been perfected in the last ten years to a high degree. They find their greatest usefulness in the acute surgical emergencies in the poor risk patients, and in injuries of the head, chest and abdomen.

These are the actual choices of anesthetics for the civilian casualty in peace time: *First, intracranial injuries:* If the patient is in poor condition, regional anesthesia is used supplemented with a $2\frac{1}{2}$ per cent solution of pentothal sodium intravenously with oxygen. In better risk patients, intratracheal ether and oxygen are employed. *Second,* in injuries involving the respiratory tract and oral cavity, including chest injuries and upper extremity surgery, intratracheal cyclopropane is used with carbon dioxide absorption. In poor risk cases, regional anesthesia and oxygen is employed. For abdominal and lower extremity operations, in good risk patients, spinal anesthesia is the anesthetic of choice. In border-line risk patients, balanced spinal anesthesia is preferred. By balanced spinal anesthesia is meant a combination of well chosen premedicating drugs followed by a minimum dose of spinal agents supplemented by a low concentration of cyclopropane with oxygen. This combination preserves, as nearly as possible, the normal physiological reactions of the patient and eliminates the danger of large doses of spinal agents. In the bad risk patient, when spinal anesthesia is contraindicated, small doses of premedicating drugs followed by gas-oxygen with or without regional anesthesia are used. This prevents further depression of the patient's vital functions.

We now come to consideration of the civilian casualties in war. In general, they are treated in the same manner as the civilian casualty in peace time. However, when catastrophes of any magnitude occur, it is necessary to handle large groups of casualties with limited personnel and equipment. This requires preliminary organization well in advance of the anticipated event. Morphia and barbiturates will be given at first aid stations. Cases of gas poisoning with irritation of the lungs will of necessity require regional anesthesia or 2½ per cent solution of pentothal sodium intravenously with the administration of oxygen for respiratory difficulties. No volatile inhalation anesthetic will be used in these cases.

Anesthesia for the wounded soldier presents a number of very complex problems. To meet these problems, too few physicians have been trained in the more improved methods of anesthesia. However, the army and navy are taking vigorous steps to overcome this need. At the Naval hospitals physician anesthetists have been organizing anesthesia along the lines of their experience in civilian practice. For the treatment of naval personnel in the sick bay itself, the risks of explosion and the lack of help narrow the choice of anesthetics to nitrous oxide, pentothal, regional and spinal anesthesia. The Surgeon General's office is now introducing the fundamental policies and organization as outlined by Tyne, Nichols and Wiggin. Specialists in anesthesia have been assigned to the various army hospitals and units to train a personnel for the care of military casualties. Others have been assigned for further specialized training at graduate teaching centers.

These are the procedures to be carried out for pain relief at the various stations of evacuation in the field of operation. At the battalion aid and collection stations morphia and rapid acting barbiturates will be administered for pain relief by medical officers. In the surgical trailor hospitals associated with the surgical hospital, the treatment of shock will be instituted by the anesthetist in charge. Minor emergency operations will be performed with intravenous pentothal sodium and nitrous-oxide-oxygen with a portable carbon dioxide absorption apparatus. Spinal anesthesia may be used at this unit if a move is not anticipated within twelve hours. At the evacuation hospital, where the major part of the surgery will be carried out, cases of shock will receive the attention of the physician anesthetist in charge, and here the patient will be given essentially the same anesthetics and methods of introduction which are employed in the general hospital, including preoperative and post-

operative care. At these hospitals will be stationed the neuro-
surgical, plastic maxillofacial, and thoracic surgical teams with a
physician anesthetist in charge of each one. The physician anesthetist
will be selected according to his ability. For example, an anesthetist
proficient in intratracheal anesthesia will be assigned to the thoracic
surgical outfit. In many cases one officer anesthetist may have to
supervise several operations at the same time. In such an event he
will depend on especially trained anesthesia assistants.

The general hospital in the zone of the interior is the final unit
in the line of evacuation to which the wounded will be finally
evacuated. Here the same facilities for anesthesia which exist during
peace time will be available.

There is a good deal of controversy regarding the ideal anes-
thetic. The variations in the human organism are so multiple that
it is still a remote possibility. An anesthetic is as safe as the anes-
thetist. The safest anesthetic is the one with which the anesthetist
is the most familiar, and that in which he has been trained. The
patient in anesthesia has been the forgotten man and there has
been committed too frequently the error of fitting the patient to
the anesthetic rather than the anesthetic to the patient. More and
more must the well trained anesthetist be a practicing physiologist.
Only in this way can he care for the varied needs of the patient.

We have in the wounded soldier a patient who is different from
the civilian in that he is robust but on the other hand under a very
considerable degree of psychic tension. His condition may be com-
plicated with alcohol, tobacco or a full stomach. He may be in
varying degree of shock for which there is need of special care
without the facilities of civilian practice. The treatment of shock
is the first consideration. Then, in the choice of drugs, morphia will
be used for relief of pain on the battlefield where anesthetics are not
available. Scopolamine would add to the efficacy of morphia under
these circumstances. It was discovered in the last war that the
majority of the wounded soldiers could tolerate much larger doses
of morphia than the civilian patient in peace time because of their
rugged physical condition.

Barbiturates will be used to allay shock, to relieve fear and
apprehension, in the field of action and also for premedication and
intravenously at the surgical hospitals. Chloroform will be used
only under the most adverse circumstances and then only by the
expert anesthetist. In the hands of the inexperienced sudden death
may occur. The same applies to ethyl chloride. However, in the

hands of the physician anesthetist, familiar with its use, it is a fairly safe agent for the induction of ether, or for short surgical procedures. *Ether is the safest anesthetic in the hands of the occasional anesthetist. It has the widest margin of safety and applicability.* Its disadvantages as stated by Kaye, of the Australian Imperial Forces in the Middle East, are concerned with the difficulty of induction, the need for increased personnel, more prolonged postoperative care, and the *incidence of respiratory tract irritation, nausea and vomiting.*

Nitrous oxide will be most useful for short operations and as preliminary to the induction of ether. The handicap of heavy cylinders and its manufacture makes it difficult to transport and to obtain. To produce complete relaxation of the abdomen nitrous oxide is not sufficient without adding ether. According to the Bureau of Mines this mixture is more explosive than cyclopropane.

According to Lundy the quick acting barbiturates administered intravenously will be used more often than any other type of anesthetic. Pentothal sodium in 2½ per cent solution should be used only by experienced physician anesthetists. Induction is rapid and the recovery early, making it of great value in this fast moving type of warfare. Rectal avertin will find but little use in this type of surgery except as the civilian general hospital. The use of cyclopropane-oxygen will be limited to the evacuation and base hospitals because of its explosive properties. This gas mixture is unsurpassed as a supplement to spinal anesthesia because of its high oxygen content. For the same reason it is unequaled as an anesthetic in thoracic operations. Regional anesthesia, either local infiltration with novocaine or plexus block, will be used wherever possible to alleviate the necessity of rendering the patient unconscious when this would react badly upon his general condition. Kaye reports that the wounded patient does not prefer it and that it makes operating difficult for the surgeon. However, if it is indicated the psychic factors of the patient can be controlled by intravenous barbiturates and gas and oxygen. There is no comparison between the use of spinal anesthesia in World War I and its present status.

Among the U. S. forces in this war *spinal anesthesia will be* used more frequently than any other method in operations below the diaphragm. The agents will be limited to novocaine and pontocaine. *It will be of great benefit to both the patient and the surgeon* in war because of its quick induction, the complete relaxation which it affords, the absence of interference with laryngeal reflexes, the

low incidence of nausea and vomiting and the early recovery with very low morbidity postoperatively. Even the patient in shock can tolerate a low dosage of novocaine and pontocaine for operations below the hips. It may be found feasible in severe injuries to the lower extremities to administer spinal anesthesia to patients so injured for relief of his pain and shock even though operation will not be performed immediately. There is the added advantage of spinal anesthesia in that one medical anesthetist may administer it to several patients and supervise them all with the aid of trained technicians.

In conclusion, there has been presented a plan for anesthesia for the surgery of trauma for the civilian casualty in peace and in war and for the wounded soldier.

REFERENCES

LUNDY, JOHN S. Anesthesia in war surgery. *War Med.*, 2: 193, 1942.

TYNES, ACHILLES S., NICHOL, WILLIAM W. and WIGGIN, SIDNEY C. *War Med.*, 1: 789, 1941.

KAYE, GEOFFREY. Impressions of anesthesia in a military base hospital. *Anesthesiology*, 3: 247–258, 1942.

DISCUSSION

GUSTAV F. BERG (Pittsburgh, Pa.): There is one type of anesthesia that I should also like to recommend to you, and that is spinal anesthesia. I have been using spinal anesthesia since 1916 and I think it is one of the greatest anesthetics we have ever had. I will not discuss sodium pentothal because Dr. Wagner knows a great deal more about that than I do.

J. HUBER WAGNER (Pittsburgh, Pa.): I enjoyed Dr. Wiggin's paper very much and think it gives us all a lot of food for thought. One statement is quoted from Lundy, that he thinks the coming anesthetic in wartime injuries will probably be sodium pentothal. I believe that when we become better acquainted with this drug, we will use it more. I might say that in the past four and a half years we have used it rather exclusively in our clinic at Pittsburgh, having over 7,000 cases, and as yet have not had even one postoperative pneumonia.

Lundy says it is good for short anesthetics. I would like to disagree with him there. We have used it in some cases as long as two and a half and three hours, using a maximum amount on one occasion of some 40 gr., with the patient recovering.

I believe that not only physician anesthetists have to be trained in this, but I think our female anesthetists as well can be trained so that they can give it without any danger to the patient, and I would ask your indulgence in the use of pentothal for your patients in the future.

BELFORD S. LESTER (Birmingham, Ala.): May I ask Dr. Wiggin about the radiating pains that follow in certain cases of spinal anesthesia? It does not seem to make very much difference whether those are administered in the army or the naval hospitals. They bother the patient from two to six, eight and ten weeks after the spinal anesthesia has been administered.

Secondly, how about the toxic hepatitis after a cyclopropane anesthesia that comes on anywhere from eight to ten days after?

SIDNEY C. WIGGIN (closing): I hold no brief against ethylene except to say that the theoretical proportions of oxygen with the gases are 90 and 10 for nitrous oxygen, 80 and 20 for ethylene, and 15 per cent cyclopropane and 85 per cent oxygen. In our experience, the cyclopropane, I think, has proved better than ethylene, although ethylene was a great improvement over nitrous oxide.

Dr. Wagner referred to female anesthetists. I suppose he means nurses. I know that the Army is training some nurses in intravenous anesthesia, but I believe that there is an element of danger, because when a patient goes bad with intravenous anesthesia, it really requires the attention of an expert anesthetist.

Regarding the radiating pains that Dr. Lester mentioned, which occur after spinal anesthesia in both legs two, four, six and eight weeks postoperatively, I have not had that complaint among my patients and I have used it since 1928.

As for the toxic hepatitis, I have used cyclopropane for the better part of ten years and I cannot recall any complication of that nature among my patients, and I have not heard it reported.

SUBTROCHANTERIC FRACTURES OF THE FEMUR*

AN OPERATIVE APPROACH FOR OPEN FIXATION

JOHN A. CALDWELL, M.D.

Professor of Clinical Surgery, College of Medicine, University of Cincinnati; Director
of Fracture Service, Cincinnati General Hospital

CINCINNATI, OHIO

SUBTROCHANTERIC fracture of the femur is one of the major problems in the management of fractures. It is most commonly due to direct violence and is distinctly an accident of middle age and late life. In the series which I have analyzed there are forty-two cases. Of these there was only one in each of the first, second and third decades, while there were thirty-nine past forty years of age. The distribution of age was. as follows:

Age	No. of Cases
1–10	1
10–20	1
20–30	1
30–40	8
40–50	8
50–60	4
60–70	7
70–80	8
80+	4
Total	42

These figures indicate that this fracture is an accident in which the passing of youthful agility is a large factor.

The portion of the femur in which the fracture occurs is encased in a large number of muscles, each of which is attached to the femur at one end and exerts some measure of control over the movement and fixation of this bone. The multiplicity of these muscles and the varied directions of their pull account for the marked displacement of major fragments as well as for the bizarre scattering of smaller pieces of bone. Because of the many small muscles which pass in a more or less transverse direction between the pelvis and the femur, and the proneness of fracture lines to be oblique or fragments to be

comminuted, it is easy for muscle to become interposed between fragments and thus interfere with reduction and delay union.

The characteristic displacement of fragments and its mechanism
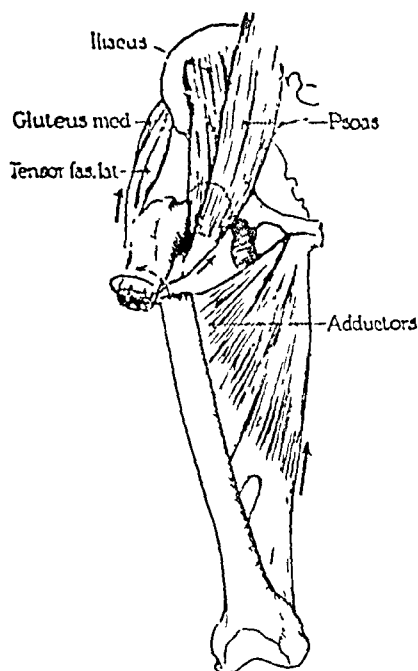
Fig. 1. Diagram showing displacing effect of muscles in fracture at junction of upper and middle thirds. (From Magnuson's Fractures. Philadelphia, J. B. Lippincott Co. Drawn by W. C. Shepard.)

of production is as follows: The proximal fragment lies anterior and lateral to the distal one, having been rotated outward and flexed by the psoas and illiacus muscles and abducted by the tensor fascia lata and gluteus medius muscle. The adductors draw the distal fragment inward. I know of no better illustration of this than the one from Paul Magnuson's book on fractures. The upper fragment is so short that it is not possible to influence its direction by leverage or to rotate and fix it. Consequently, it is necessary to place the distal fragment in line with the proximal one. Where the line of fracture is transverse the fragments may occasionally be engaged by manipulation under general anesthesia, but when the line of fracture is oblique or the fragments are comminuted, reduction is rarely possible and position of fragments must be maintained by traction. Some form of balanced traction is generally advocated in which the leg is

rotated externally, the thigh is flexed and abducted, and the knee is flexed to relax the hamstring muscles. Russell's traction or suspension under a Balkan frame with traction by means of a Thomas splint with the Pearson attachment are methods most commonly used.
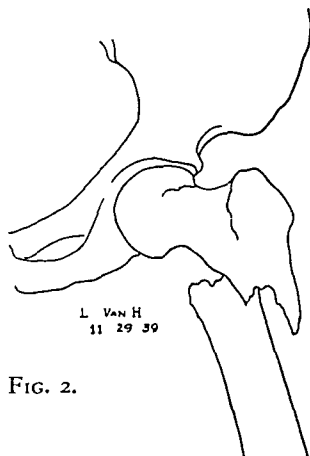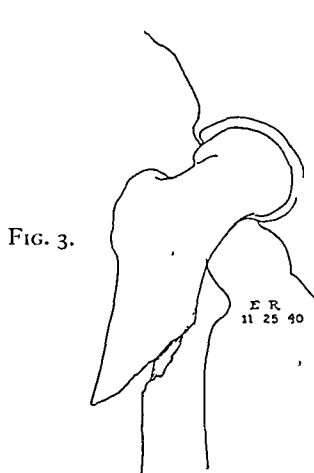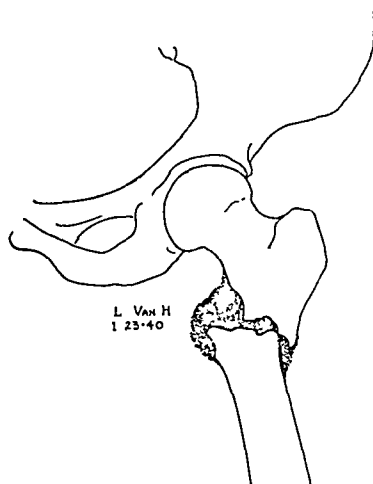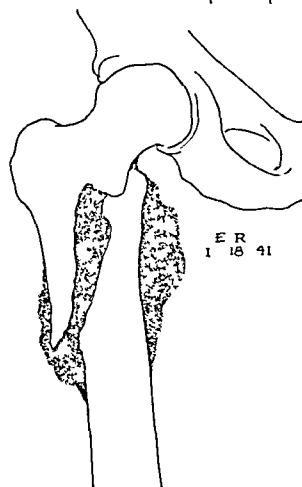
FIG. 2. Usual displacement and result after callus had formed. (Tracing from x-ray.)
FIG. 3. Position during traction and final union. (Tracing from x-ray.)

When traction fails to effect a satisfactory reduction there will be a high incidence of delay in union, angulation and shortening. In such cases we believe that many of these breaks should be subjected to open fixation at an early date. Formerly, we were inclined to temporize because of the magnitude of the operation, but with the approach, which I shall describe, our attitude toward the problem

has been considerably altered, even though the fracture is frequently associated with such unfavorable factors as senility and obesity.

The common approaches are (1) the anterior one described by
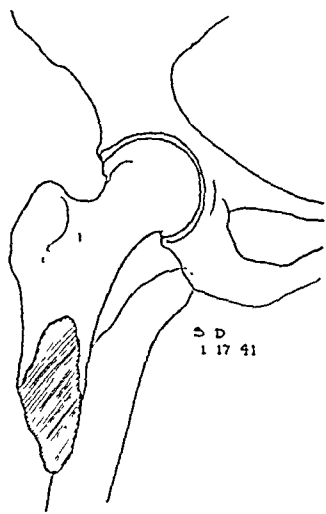
FIG. 4. Position during trac-
tion. (Tracing from x-ray.)
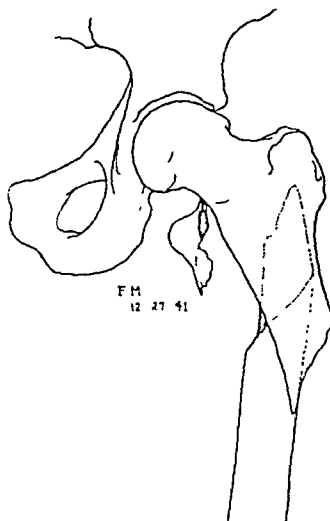
FIG. 5. Position during trac-
tion. (Tracing from x-ray.)
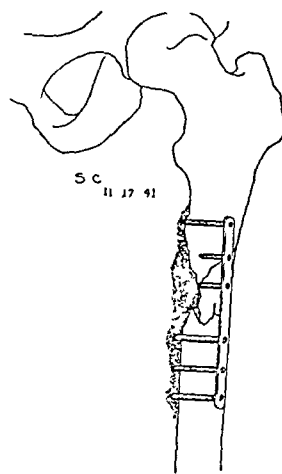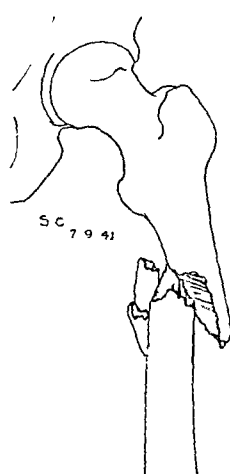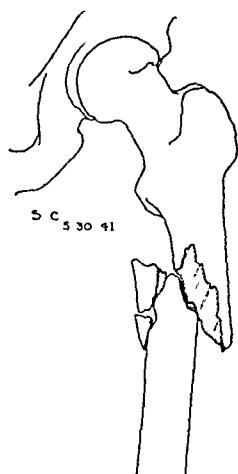
FIG. 6. Position and result when operation was performed late. (Tracing from x-ray.)

Henry in which the incision follows a line from the anterior superior spine of the ilium to the outer border of the patella, and (2) the lateral one in which the incision extends downward from the great trochanter. In both of these the bone lies very deep and must be discovered through the fibers of the vastus lateralis muscle. I know of few more harrassing operative procedures than trying to expose

and mobilize fragments of the upper end of the femur in a muscular patient through either a lateral or anterior approach.

The approach which we have employed in six cases has made the
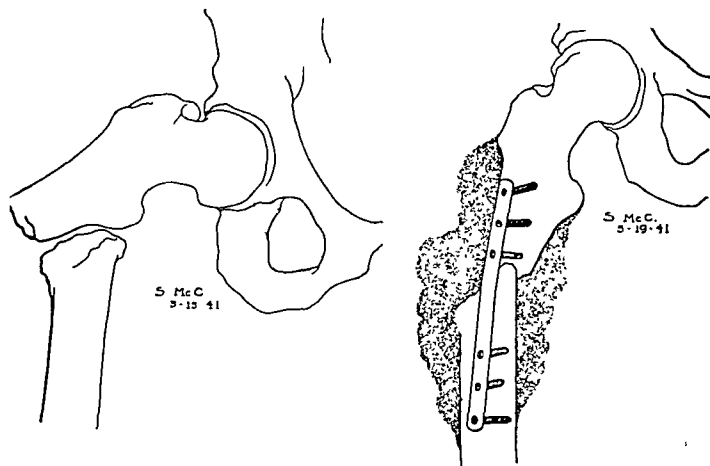
FIG. 7. Position and result when operation was performed late. (Tracing from x-ray.)
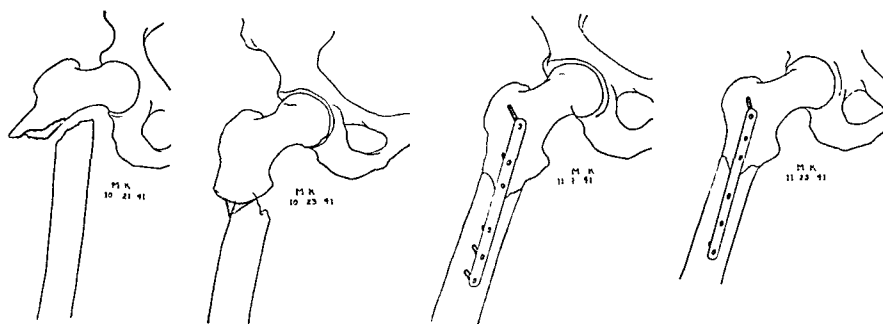
FIG. 8. Result when operation was performed early. (Tracing from x-ray.)

operation much easier for the reason that the muscles can be separated, the bone is nearer the surface and structures which must be avoided, particularly the sciatic nerve and femoral vessels, are not so placed as to cause one to fear their injury. This operative approach is not original with me, but I do not know to whom to give the credit.

When possible we like to apply a single hip spica to the patient several days before operation. When doing this we place over the operative field several gauze pads about the size and extent of those to be used as wound dressings after the operation. The case is bivalved before it is quite firm and is laid aside to harden and be ready for application at the completion of the operation. By this plan

the anesthesia is not prolonged by the period necessary to apply the cast, and the operation can be performed on an ordinary operating table.
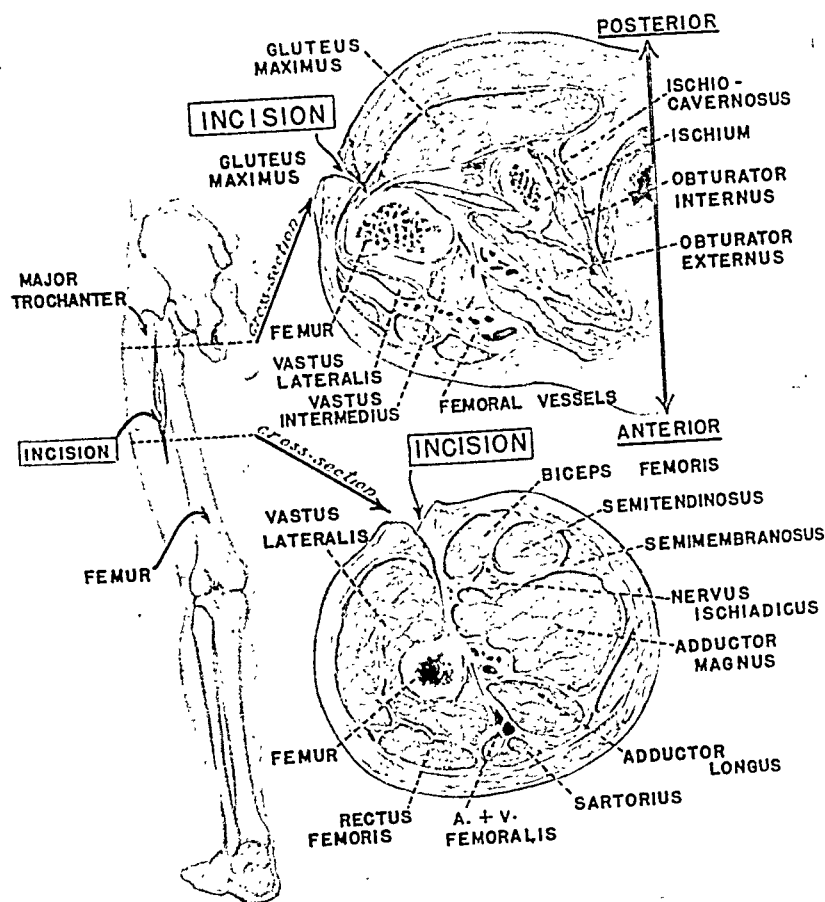
FIG. 9. Cross sections of thigh at levels of trochanter and upper third showing approach to bone by separation of vastus lateralis and biceps femoris. (Adapted from Manual of Surgical Anatomy. Medical Department of U. S. Army and Navy.)

The patient is placed face down on the table and the side to be operated upon is elevated about two inches. The incision follows the posterior border of the fascia lata and begins at the greater trochanter and extends downward eight to ten inches. In its upper portion the insertion of the gluteus maximus muscle into the fascia lata is cut, leaving enough of the insertion to repair after completion of the operation. In the distal portion of the incision after the insertion of the gluteus maximus muscle is divided, the biceps femoris,

which lies on the medial side, can easily be separated from the vastus lateralis by blunt dissection. The femur is then felt at the bottom of the incision. From above downward the attachment of the
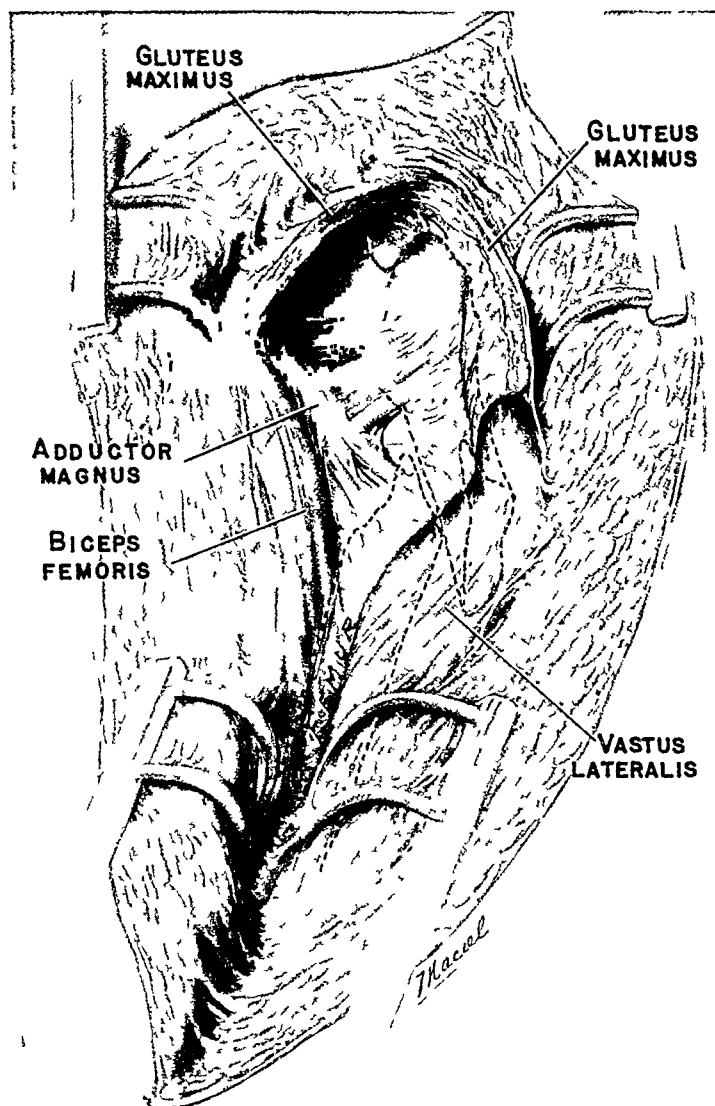
FIG 10 Drawing showing operative approach

quadratus femoris and then that of the adductor magnus muscles may have to be separated from the posterior surface of the femur. The proximal fragment usually lies anterior and external to the distal one. Each must be separated from any attachments it may

have formed, then by levering or clamping with bone holding forceps and manipulating, the two fragments are maneuvered into suitable end-on apposition. The method of permanent fixation is a matter of choice. In all of our cases we have used six-hole plates.

Silk is used for ligatures and sutures throughout the operation. After completion of the operation and dressing, the posterior shell of the cast is laid on the patient and he is turned and lifted in this case to stretcher or bed. The anterior shell is then applied and the two are strapped together accurately with adhesive straps.

In all of the six patients operated upon union has been prompt and firm and good function has been restored.

Two of our cases did not come to us until efforts by others had been ineffectual and considerable time had been lost and in two of our own cases we continued traction methods beyond a reasonable period. In the other two cases operation was performed within ten days, and in both of these good useful function was restored in four months. The periods of hospitalization were fifty-nine days and sixty-four days, during which time a cast was in place eight weeks and seven weeks.

Of the thirty-six patients treated without operation both Russell's traction and Thomas splint with Pearson attachment were used; in several cases both were tried. In all cases the traction was followed by the application of a cast. Two patients died during the treatment. In the thirty-four survivors union resulted in all, but two were unable to bear weight before eight months. The maximum shortening was one and one-half inches in two cases, one inch in two, one-half inch in four, and none in twenty-six. All have united by excessive callus.

SUMMARY

The posterior approach offers a much easier and simplified method of operating upon subtrochanteric fractures of the femur. In suitable cases, when a satisfactory reduction cannot be obtained by other methods, we believe that an early use of this procedure will be followed by better results, less inconvenience to the patients and a shorter convalescence.

DISCUSSION

HENRY C. MARBLE (Boston, Mass.): I am very glad that Dr. John Caldwell brought up the subject and I think if we had more confessions of defeat, that it might be the ground for discussion. I do not recall that I

have heard that discussed very much, particularly in wartime. My recollection of that particular injury which Dr. Caldwell brings to our attention as a defeat, is of a through-and-through wound that passes through the soldier's thigh; he sustains a subtrochanteric fracture with comminution and you are confronted with two or three problems. The first problem is, what are you going to do to hold the proximal fragment? The first picture Dr. Caldwell showed by Dr. Magnuson is very simple, but when you have overcome the degree of upward and outward displacement of the proximal fragment and just at the time that you have got the distal fragment to meet it, the proximal fragment goes a little further, and when you get that it goes up still little further. When you end up, you have the patient literally in a knee-chest position in an effort to hold it.

I have never seen one of those cases handled by plaster of Paris fixation, such as is now used, and I wonder how it would manage that proximal fragment. That is always a problem and I recall having some of these men treated with their knees in flexion, their thighs in flexion or better, and dressing the wound on the front of the thigh, and then lying down on the bed and dressing an equally large and sometimes more infected wound in the buttocks. It is a difficult fracture to handle.

There is one other factor in the compound injuries which I think we should bear in mind that this is a source of secondary hemorrhage. There is a large artery which accompanies the sciatic nerve, which is difficult to control, difficult to expose, and having exposed it, difficult to tie. It is also intimate to the sciatic nerve and I have often wondered how many of these cases have bled and the sciatic nerve has been tied with the artery in an effort to control the hemorrhage.

I think this is a very important matter for discussion and I am very glad Dr. Caldwell brought it up.

FRANK P. STRICKLER (Louisville, Ky.): I think Dr. Caldwell has given us a very timely paper and has shown us some excellent results. When Dr. Morrison asked me to discuss this paper, I ran through a series of 100 cases that we had had, and I found essentially the same facts that the doctor has found, namely, that this is an injury of middle life and of late life; but I also found, much to my surprise, that in these injuries under thirty years of age, they were complicated by compound fractures of the tibia and fibula, and these cases occurred in automobile accidents. Of course, a compound fracture of the tibia and fibula complicates this injury considerably.

We have operated upon only 10 per cent of our patients. We use the lateral incision from the greater trochanter down parallel with the femur, and we have done our cases on a hollow table. We seem to think that we can pull it in line better that way and hold it in position that way.

The only trouble that we have had with our cases is that we have found that a large number of them have been rather severely comminuted, and that would make a plating operation rather difficult due to the fact that we simply had nothing for the screws to hold to.

We have used largely conservative treatment, and with very good results. We have used the Thomas splint with the Pearson attachment, and a Kirschner wire through the crest of the tibia just below the tibial tubercle. We put that through under local anesthesia, as a rule, in bed, without moving the patient to the operating room at all. We then apply a Thomas splint that has a rather snug fitting ring. We believe that the ring helps hold the upper fragment and helps to maintain position. We put the extremity up in slight flexion at the knee. This relaxes the hamstring muscle and the flexor muscles. We then apply a weight. I think if you are careful in applying this weight and if you watch your cases carefully with x-rays and measurements, you will have no difficulty.

It is very easy to measure these cases, and we x-ray our patients not less than every two weeks, and if we have any difficulty with them, we x-ray them more frequently than that. I think one of the reasons many of us get into difficulty with fractures is that we are too Scotch with x-rays and we do not take enough of them. That is really the only way in which you can tell what a fracture has done. Of course, you do not want to go to the other extreme and x-ray them so much that there is a possibility of interfering with bone growth. That is the other extreme, but I believe, from my experience at least, that it is safe to x-ray a patient at least every two or three weeks, and that by watching these cases carefully, you can get good results.

I do not know why, but I have not had a great deal of trouble with this fracture and I have gotten very good results. We have had no cases of nonunion and we have had very good functional results.

As far as the incision goes, I can find no fault with the doctor's incision at all. The only objection that I see, or criticism that anyone could offer to this type of incision, is the position and the possibility of its becoming contaminated when the patient eliminates. That is the only criticism that I could offer to it, because we all do have accidents and get incisions contaminated in that region.

I recently had a case that was very interesting to me from this standpoint. Of course, it might not interest you much, but my father saw this man with a fracture of the type that we are talking about, forty years ago. The man fell about a week ago and refractured that femur through the same site, and he came to me forty years later with a fracture through exactly the same fracture that he had years previously.

ROBERT P. DOBBIE (Buffalo, N. Y.): My experience with this type of fracture has been very limited and I really have no right to discuss Dr. Caldwell's paper. All I have to say is that my experience has veen very limited in handling this fracture and I have had my difficulties. Unlike Dr. Strickler, I found it a very difficult fracture to handle in the few that I have handled, and as usual, Dr. Caldwell brings a point that is practical and concise and very valuable. The posterior approach is very intriguing and I am sure I will keep that in mind with the next fracture of this type

that I have to treat, due to the difficulty I have had in my few cases in getting through these large muscles in these very obese patients.

FRED H. SMITH (New York, N. Y.): In many of the subtrochanteric fractures that we have had, we have attempted to fix them by plates, and in some of the very high ones even use a Smith-Petersen nail with a Thornton plate. The purpose of internal fixation is, in the first place, to get anatomical position with rigid fixation which permits moving the patient and allows him to get up. If we get firm, rigid fixation, we believe it is not necessary to use any plaster of Paris afterward, and the patients get a much quicker and a better return of function.

After using a plate—we usually use a six-screw plate—we use one or two hand-fixing screws which are put in at a different angle from the screws holding the plate, and in that means it is not possible to bend or twist the plate at the fracture line. We believe we can start hip motion right from the start, and we also believe the patients can be allowed up in a chair in a short time, and that cuts down very greatly on the possibility of postoperative pneumonia and other conditions that these older people get.

H. GURTH PRETTY (Montreal, Canada): This subject of subtrochanteric fractures, I think, is a very interesting one. It has given a good deal of trouble in Montreal. We run into a large group of them, and our experience has been that by the closed method, we were not able to get a reduction; we got a certain degree of shortening and deformity and we had a long convalescence. Furthermore, lately we have been running into a fair group of compound fractures, and we have found in these compound fractures that our difficulties are even more severe than before, so that on some of them we have been attempting almost immediate plating. In other words, the wound had been débrided, it has been watched closely for a period of a week, with sulfonamides, and using a. t. s. and also anti-gas gangrene serum at the same time; then we have gone in and reopened the wound and replated it. The point I want to take up in connection with this slide is that this was a man of about fifty who was struck by a steel rail. He had a hole in the side of his thigh big enough to incorporate two fists, and no matter how we tried to manipulate him, this somewhat bizarre "butterfly" got in the way and our pieces flew all over the place.

The second thing was the question of holding this bone in position for plating by using some lash screws and six-screw plates, and I found by using a small plate anteriorly with the six-screw plate on the side, we seemed to get a very much better position than by a single plate and lash screws.

GUSTAV F. BERG (Pittsburgh, Pa.): I think that Dr. Caldwell is to be congratulated for bringing before us one of the most difficult fractures that we have to contend with, and I believe that Dr. Caldwell has stressed a point which is very valuable. This is one of the types of fracture that, in my opinion, is always an open reduction case.

There is one thing that you must remember. When you are getting up into the trochanteric region, the bone is soft and you may have difficulty

with your screws having sufficient purchase. In these comminuted fractures it is often a good idea to go back to Dr. John Murphy's theory, doing an intermedullary graft, taking a piece of bone, cleaning out the trochanterous structure about, putting in the bone and putting on your bone plate.

HENRY C. MARBLE (Boston, Mass.): I think there is one matter that we have overlooked. I do not know that it was overlooked, but we always believe, also, that fracture in this particular location is very apt to be metastatic and it is a pathological factor. This seems to be a point of election for certain metastatic cancers, Paget's disease, and we must not forget that in our discussions.

JOHN A. CALDWELL (closing): I am very grateful to Dr. Smith for the suggestion for the management of these cases. Further than that, I think there has been very little discussion of the procedure. Most of the men have given very good reasons for it, which is lack of experience. The purpose of this was to try to popularize experience with it.

It might carry a little further conviction if I would give here a report of one of these operations. This was done by the resident surgeon on the service, a man of extraordinary capability who does everything well, and who writes this report. This was the ordinary, routine, operative report which I find it very difficult to improve on in describing the operation:

"The entire leg was then encased in a double layer of sterile stockinette which was extended as far as possible upward on the posterior aspect of the thigh. The draping was supplemented with sterile towels and with plain sheets in such a manner that the leg was within the field of operation and available for manipulation." That is, the leg, covered with the sterile stockinette, was outside all sheet draping so that it was possible to take up the entire leg without contaminating the field at all.

"A longitudinal incision was made after opening the stockinette in the palpable groove between the vastus lateralis muscle and the biceps femoris. The patient was fairly obese, and about $1\frac{1}{2}$ inches of adipose tissue was cut through before exposing the deep fascia. The approach came directly down on the tendonous insertion of the gluteus maximus muscle, and the lower portion of this was divided close to its insertion. Then by separation between the belly of the vastus lateralis and the biceps femoris, the shaft of the femur was readily exposed without exposure or injury to any major nerves or vessels. The muscle fibers attached to the bone surface were stripped free from below upward to expose the fracture site and the hematoma surrounding it.

"The proximal fragment was displaced anteriorly and laterally and the distal fragment posteriorly and medially with approximately $1\frac{1}{2}$ inches of overriding. The ends of the two fragments were dissected free and grasped with Lambot bone-holding forceps, and by manipulation of these two instruments and the manipulation of the lower leg and foot by an assistant, and with the use of a smooth bone skid, the displaced bones were reapproximated in 100 per cent anatomical reduction. A five-inch Shermann plate of

stainless steel with six holes was now applied to the posterior surface of the bone, and one screw was inserted above and one below after preliminary drilling of the bone. With these screws in place, a Lowman clamp was applied around the fracture site and over the Shermann plate to maintain the two bone ends in accurate apposition while the remaining drill holes were made and the plate fixed firmly in position. Three screws were used above and three below."

That gives in very complete detail, I think, everything that is done with this, and that is about the procedure as it is carried out. I think that comparing this approach and this manipulation with the ordinary lateral and anterior approach is like comparing, as far as speed and effectiveness are concerned, the difference in travel by train and airplane. It is much more direct and much easier.

TREATMENT OF INJURIES OF THE HAND
REQUIRING SKIN GRAFTING

LIEUT. COMDR. THOMAS L. HAWKINS, M.C., U.S.N.R.

U. S. Naval Hospital

FARRAGUT, IDAHO

IT is not the author's intention to enter into the fine art of skin grafting as applied to the extensive practice of the plastic surgeons, but merely to call to the attention of surgeons engaged in the treatment of injuries, the importance of *immediate* skin graft in injuries of the hand.

Commonplace injuries of the hand are the stepchildren of surgery, in that they are by far the most frequent of injuries and are cared for by thousands of physicians and surgeons. We accept these injuries and resultant deformities usually as trivial, often because of their being commonplace, which leads to indifferent care. This paper then is a plea for a sharper focus on these injuries and a mobilization of our present surgical knowledge from all sources, toward more intelligent care.

The challenge thrown out by Walter Estel Lee's oration on Fractures and Other Traumas at the Clinical Congress of the American College of Surgeons, Boston, November, 1941, should be a whip and if so accepted may overcome the stigma of the quotation of Dr. Meeker, that, "A general surgeon is one who does not do anything particularly well." The sting should prompt the general surgeon to borrow from the vast field of knowledge of his specialist brothers, and to apply approved methods in the care of injuries of the hand. In so doing he must become a student of pathology, orthopedics, plastic surgery and neurosurgery. The proper marshalling of this knowledge will lead to real intelligence in the care of hand injuries, and the general surgeon may make for himself at least a small spot in the sun and find that he can do some *one* thing particularly well.

The hand as a member of the body has never been more important in the history of the world than at this particular time; this being a mechanical age a resultant mechanical war has ensued. The importance of the full function of the hand is obvious. We forget that we have ten digits on our two hands; we do not have an adequate conception of the value of these members or of their co-related values. A hand without digits is practically a total loss, the hand

might as well be absent. Functionally, the digits may be divided into two major correlating units, the thumb and the fingers, each probably of equal importance. A hand without the thumb is a 50 per cent loss of hand function, conversely, the loss of four fingers is a 50 per cent loss. The relatively greater value of the thumb over the individual fingers lies in its being a member in apposition to the four fingers. The loss of either of the cardinal components of the hand nullifies the palm almost entirely.

The original work of infections of the hand by Kanavel and.the continuation of this missionary work by Koch and Mason and others, have entirely revolutionized care of hand injuries and, in fact, have made an expose of fanciful claims of the value of skin antiseptics. At last the fundamental principle of skin cleanliness through the media of soap and water properly applied, has driven from the field myriads of caustic cell destroyers masquerading under the term of antiseptics, many of which were not even antiseptics, and almost all were cell destroyers.

All hand injuries should have no further first aid than a sterile dressing and a tourniquet applied to relieve obvious serious hemorrhage. Pressure control of hemorrhage at the site of the injury generally should be condemned. The injured hand, when seen by a surgeon, should be carefully inspected and tested by direction to the patient as to nerve, tendon and bone injuries. X-rays should be taken in practically all but the most simple of lacerations. Fractures are of secondary importance, in that lacerations and soft tissue damage demand first consideration.

The hand should be thoroughly cleansed with soap and the use of gallons of water. Injuries on the palmar surface require a brush; others should be cleansed gently with sterile gauze. In injuries of the fingers, nerve block anesthesia should be employed before careful cleansing of the wound area is instituted. Larger, more complicated injuries of the hand require general anesthesia of a preferred type. No injuries are *ever* repaired with local procaine infiltration. Ideally, all hand injuries should be accorded operating room asepsis. Practically this may not be obtainable, nevertheless, this goal should be set up for future attainments. No surgeon would permit general abdominal exposure without proper masking and aseptic procedure, yet many a hand injury presents far more inviting fields for expired air contamination.

A sterile rubber glove is fitted on the hand, an incision made over the desired area (rubber shortage may preclude the use of this

extravagance) and sterile rolled stockinette used. Hemostasis is essential to correct repair. The skin edges are removed in an intact circle which should include the walls to the bottom of the laceration. Débridement should be thorough but not reckless. The use of powdered sulfanilamide sprinkled lightly over the denuded area seems to be definitely beneficial; however, the dumping of large amounts is certainly detrimental to clean healing. Any type of fine nonabsorbant suture may be employed, and a stitch used which does two things: first, co-apts the skin edges in gentle approximation, and second, does not exert cutting pressure.

By far the greatest number of injuries to the hand are to the fingers and thumb and need not be enumerated. The injuries which require immediate skin grafting are those with extensive loss of skin, usually through crushing injuries and burns. The simplest of these are the slicing lacerations of the finger tips, commonly occurring in contact with jointers, meat cutters and other mechanical cutting devices. When the tip has been excised with the greater portion of the ball of the finger and the distal phalanx exposed, the area should be grafted. These injuries, if not grafted with skin, leave the peculiar toothpick finger which is permanently damaged, particularly for fine work. Close observation of the function of these deformed fingers will show how little they are used. The author has long since ceased to scoff at the patient who brings in the severed portion of the finger. Usually if seen within a short time careful cleansing and application of the severed segment will result in a take.

The wringer-type of hand injury in which the hand has been literally peeled of the skin, requires meticulous care; not infrequently the skin is not lost at all, simply rolled up on the hand. All of these are hospital cases unless operating room facilities are available in the office or clinic. If seen within a few hours the rolled skin should never be sacrificed, no matter how devitalized it may appear. After careful cleansing of the hand with soap and water, and no antiseptics, the surgeon should assume the rôle of the plastic surgeon and proceed to apply these principles in "grafting" the denuded area. It is not infrequent that repair of these injuries may require not minutes but hours. Final results will more than justify the fastidiousness of the surgeon. Should skin be lost, the surgeon should immediately replace it with free grafts.

The most ideal is the full thickness graft; however, this leads to delayed donor area healing so that the thick split graft may be advisable in some cases. This, however, does compromise the ideal

full thickness graft. The extensive hand injury will require a longer operative time than most major abdominal procedures and justly so, for the permanent disability may be far reaching.

While the wringer-type of hand injury with skin peeling provides the ideal injury for immediate skin grafting, similar injuries, such as result from automobile accidents, with extensive soft tissue and deep structure injuries, lend themselves to immediate grafting for skin losses. Here again basic cleansing principles must be adhered to before repair may be instituted.

Any surgeon who presumes to accept responsibility for the care of these injuries must be adept in nerve, tendon and bone surgery, and must, therefore, keep himself familiar with current advances in these particular fields. It should be quite evident that a neurosurgeon, an orthopedist and a plastic surgeon would be a crowd in the operating room. It should be emphasized that even in these ragged, lacerating injuries, radical débridement should be frowned upon; every available milometer of skin should be saved, even at the expense of long operating time. By careful fitting and key suturing many denuded areas may be covered. Palmar surface skin is frequently lost because of failure to realize the increased thickness of the skin in this area so that sutures are placed superficially. After the slough of the thick cornified area, the suture may not even have penetrated the thickness of the skin, hence, lack of co-aptation and resultant poor feeder circulation. Denuded tendons may be grafted with confidence of success, but should have full thickness grafts and careful attention to proper pressure dressings.

The interposing of fat, or the cutting of the graft with fat to prevent adhesions between the graft and the tendons, is an excellent method to insure its death except for pedicle flaps. Fat does not prevent adhesions; on the other hand, it is an excellent insulating tissue against good blood supply to the base.

The use of mechanics waste as a pressure medium, as popularized by James Barrett Brown, is infinitely satisfactory, provided the dressing is applied carefully so that it will function. It certainly has no magic inherent powers to apply uniform pressure unless equally distributed pressure is applied through the application of the dressing. Once the dressing has been applied and examined daily to determine whether it is functioning properly, it should be left alone for a week. Frequent dressings only increase the hazard of infections; the gentlest change is traumatic to delicate healing structures. Curiosity to view grafts should be repressed.

It is essential that these injured hands be splinted in complete rest. Many a two-hour painstaking operation is ruined in a few minutes by the patient struggling in postanesthesia. If proper, adequate, light splinting material is not satisfactory, plaster should be used which later may be shelled for observation and dressings.

The surgeon who cares for these hand injuries should acquaint himself with the armamentarium of the plastic surgeon, so that he may have adequate and applicable instruments, otherwise he may find himself a boilermaker in a watchmaker's shop. Careful, gentle and meticulous handling of tissues, to a point of being ultrafastidious should be the guide posts of any surgeon in the care of hand injuries. Time taken in the care of these injuries should be of no importance.

Some time has been consumed in detailing the hand cleansing as it is so vital if grafting is necessary. As a matter of fact, the method of cleansing of the hand may quite definitely determine success or failure of the graft regardless of how perfect the graft may be cut and applied.

Full thickness skin loss in burns lend themselves admirably to early grafting and here again, basic cleansing principles apply possibly more forcibly than in lacerated wounds. After all, a burn is a wide open wound and should be treated as such. As soon as a clean base is established split grafts should be applied. One thorough and careful operative procedure at this time may save numerous later operations which frequently are basically palliative. The dermatone makes available an accurate device for skin splitting so that there is no excuse for delay in grafting. It has been pointed out in treatment of burns, even if the graft is a failure, it has supplied an ideal dressing and to some extent has prevented contracture.

The potentialities of permanent disability with economic loss in injuries of the hands may far surpass potentialities in so-called major surgery elsewhere.

Treatment of injuries of the hand demands far more consideration, far more sober thought to combat permanent disabilities. These injuries are worth most considerate, thorough and gentle care and should be elevated in importance to the sphere of major surgery. They should also be recipients of the fruits of advances in specialized lines of surgery. Possibly it is unfortunate that the largest percentage of them are destined to be cared for by the general surgeon, but with wisdom this garden variety of injuries may be the vehicle to carry him where he may do many things well.

DISCUSSION

EARL C. PADGETT (Kansas City, Mo.): Dr. Hawkins has, we all agree, brought up a subject that is very timely. He has tried to lay down general rules for the care of hand injuries and as in all cases in which one lays down general rules, there are many exceptions which one might use with advantage. I will not go over those particular things, only to say that sometimes I would think that possibly fractures might not be of secondary importance.

Although one might lay down a general rule that general anesthesia is always necessary, I think that possibly one sees many cases in which local anesthesia might be used with some good judgment.

I have heard many men state that the completely severed portion of a finger should be reattached and given blood supply, but I have never seen such a thing happen if the cut was deeper than the subcutaneous tissue.

The statement was made that the most ideal graft is a full thickness skin graft. Personally, we have not used the full thickness skin graft except on web fingers in children for many years, the reason being that with full thickness skin graft, although the take is perfect and the protection is the best that any skin graft will give, you run about a 20 per cent chance of not getting a take, even on a clean surface, and if you do get a take you may get some blisters. Those do not make so much difference on the hand because one is not looking for an ideal cosmetic result, particularly, anyway. We have used what we have termed (and the term is probably bad) the three-quarter thickness skin graft, basing that designation on the fact that the graft is cut to the last quarter thickness of the skin. This graft gives good protection and the cosmetic result is good and it practically always takes.

On an aseptic field with proper fixation, dressings and so on, one can rest assured that his takes will run close to 100 per cent. Of course, on the granulating areas we do not try to use a fixed skin graft because of the liability that one may lose the graft. We usually cut the skin graft fairly thin, preferring to make the chance of getting recoverage high, and if there is contraction after the hand is healed, one can relieve the contraction.

To generalize then, on the hand we have laid down certain rules which to us seem somewhat of value: For most skin and subcutaneous tissue losses or contractions in the palm or over the dorsum of the hand or of the fingers, the application of the proper skin graft is preferable to the application of a skin flap. The exceptions to this statement are met when tendon, bone or joint is laid bare or when it is anticipated that a tendon grafting operation is to follow. Then a skin flap is definitely the indication.

On the fingers on which it is essential to cover a guillotine amputation or the original injury has been of the guillotine type, to get added length with adequate protection a pedicle skin flap is the indication. This is also the case when one side of the finger has been denuded down to the bone. When it is desirable to increase the length of an amputated digit, coverage with a skin flap will give one about one-quarter of an inch increase in length in

addition to giving a well padded protection. In my experience, a skin graft does not give sufficient protection over the end of the digit if applied immediately on the granulations springing from severed bone.

Correction of webbed fingers of either the congenital or acquired type is most efficiently done by application of thick skin graft between the fingers after complete severance of the web.

WILLIAM E. BROWNE (Boston, Mass.): I do not believe I have a single thing to add to Dr. Hawkins' paper. There is nothing we do that is materially different from what he has outlined in his paper. Perhaps we would not go quite as far as he goes in saying that fractures are not of prime importance in changing abnormalities in the skeletal framework of the hand before attempting the soft part repair. If, for example, you consider what has been said this morning about subtrochanteric fracture, about the vastus lateralis, the gluteus maximus and adductor magnus, and the danger of injury, and if you transfer that to the hand, to the thumb, for example, and the structures attached to the outer border of the proximal phalanges, that is the outer head of the phalanges and the inner border, the inner head of the flexor pollicis proximal and the oblique adductor of the thumb, to attempt a skin graft between the first and second fingers with those antagonistic muscles unattended to by lines of fractures not repaired, would result in considerable deformity as one might attempt to reconstruct the hand with soft part repair of one kind or another.

I do think that Dr. Hawkins' note on anesthesia, passed over lightly, is of importance. He has underlined in his paper, which he was kind enough to send me, the words that we never use local infiltrative anesthesia in attempting these things where other forms of anesthesia may be permitted. I do think it is unfortunate, when one sees these grafts which do not hold and when one sees in the fifth or sixth layer of the skin (removing sutures ten days rather than a week after they have been put in) that the deeper layers have not been attended to. One can avoid that very nicely, obviously, by undercutting the edges properly before grafts are put in.

I have not had the nice results with Dr. Padgett's dermatone, which everyone should have, and I have used it in other parts of the body with better results sometimes than in the hand; as I say, I have not had the results which I am sure he has had. I do think that it is important in these finger injuries, where the tips of the fingers are gone, in using little postage stamp grafts there, that thick skin grafts are advisable. I am sure it is almost impossible to talk to men who have had tremendous experience in this field, to express certain dogmatic points, except to prevent those who are not adept in handling these things from attempting to handle them. They must be done, obviously, by persons who have had considerable experience.

I think it is most important to bear in mind what Dr. Hawkins said about not wasting ruffled up and raveled up plaques of skin. Suppose the skin does not take at all which one unruffles or pulls down; there is much

better healing where one uses that skin, even though a good part of it is sacrificed later on, and in the event that full thickness graft or some other form of graft is later necessary, it will take very nicely. I think in a certain percentage of these cases—and I cannot give any figures—in which the tip of the finger is done, that it is highly desirable, although there is a good deal of atrophic disturbance, to·inject the digital nerves before a graft is placed or an inlay is laid, so that the tip of the finger, when one attempts to use it later on, will not be very painful.

I heard Dr. Lund, who was my great teacher, define minor surgery one time as surgery done by minor surgeons, and the more minor the surgeon, the more major became the surgical thing he was doing. If that is true, it certainly is true in the hands, and I would advise persons going to the other end of the ladder, to learn how to do a thyroid, to know the triangles of the neck, to be able to do a breast very thoroughly, to be able to take out a gallbladder well, to do a hernia very, very nicely, and after that, come back to the hand which seems to offer so many difficulties.

Lastly, I might be permitted to pay tribute to the President of this Association, of which I am not a member, for the very excellent work he does here in Boston on these very difficult problems involving crippled hands.

A. WILLIAM REGGIO (Boston, Mass.): I would just like to add one word on a thing that we are apt to forget in these hand cases. We speak of splinting the hands right along, but we must not forget to splint those hands in the position of rest, sometimes called the position of grasp. With relaxation, it avoids tension and is infinitely more comfortable, so that the position of grasp or relaxation, if possible, is the way that we should do our splinting every time.

In the second place, do not forget the shoulder. So many times we see these people come in who have had trouble with their hands, and at the hand clinic at the Massachusetts General Hospital, we have the habit of saying to these fellows, "Put up your arms," and there you are. The injured hand gets a glued up shoulder. Always give the instruction, on whatever you do on the hand, that the patient must put that hand up over his head at least three or four times every day. That is a point worth remembering.

JOSEPH L. BURNETT (Boston, Mass.): There is one thing in this discussion of hands that has not been mentioned and I think it is very important. Most of these patients with injured hands land in the hospital and the suturing of tendons and other things connected with compound injury of the hands are done usually by the lowliest intern on the staff. I think you men should go on record as saying that all hand injuries other than a simple lacerated wound, should be done by no one less than the resident.

THOMAS L. HAWKINS (closing): I certainly did not wish to convey the impression that the fractures were unimportant. They certainly are important and it does not do you very much good to spend a lot of time on the

soft tissue and then not take care of the fracture. On the other hand, I still believe that in the treatment of these, you may get a perfect result from a fracture standpoint, and then have a nice infection which will obviate all of your good work on the fracture.

I think we ought to remember what Dr. MacFarlane said about the use of sulfanilamide. Certainly, I think it is a grand adjunct to surgery, but just like antiseptics, it is not the really important thing. The really important thing is the careful cleansing, the débridement of the wound, and we certainly should not expect miraculous results from the use of sulfanilamide.

Remember what I said about not dumping large amounts of sulfanilamide into the wound.

Dr. Reggio's statement on physiological rest is of a great deal of importance.

INTRACAPSULAR FRACTURE OF THE NECK OF THE FEMUR TREATED BY INTERNAL FIXATION

AN ANALYSIS OF 128 CASES

RALPH G. CAROTHERS, M.D.

Attending Surgeon, Good Samaritan, St. Mary's and St. Francis Hospitals

AND

CAPT. NICHOLAS J. GIANNESTRAS, M.D.

Attending Surgeon, St. Mary's and Deaconess Hospitals

CINCINNATI, OHIO

THIS is a report of 128 consecutive cases of intracapsular fracture of the neck of the femur in which the patients were treated by one method. It comprises all but seven cases having such fractures, which either of us have seen in the seven years covered by the report. The seven cases in which pinning was not used were patients who refused operation or the patient was obviously in a dying condition when first brought to our attention. Nevertheless, among those patients operated upon, tremendous risks have often been taken on very feeble patients. Many times we have been gratified to see an individual, who at first appeared to be headed for the grave, walk out of the hospital on his own feet. Our deaths occurring in patients while under treatment have been very few, and no patient has died within forty-eight hours after operation.

PROCEDURE

The operation has been described in previous papers and only a few points need be mentioned. In our early cases, the pins were introduced in a converging manner as first described by Austin Moore. We later got away from this idea, and for approximately the last 110 cases have tried to get our pins in as nearly parallel as possible. We have also attempted to start the pins at a lower point on the shaft than we did in the beginning. For this reason, we have sometimes had to drill holes through the cortex of the shaft. There has been one other change in the operation and that is, instead of spinning the pins in, they have been driven in. We have devised an instrument consisting of a rod of steel into one of which the pin is screwed. This rod then acts as a handle, and with an ordinary hammer, the pin is driven home.

The results of this series are very gratifying. Only nine patients or 7 per cent have died while under treatment in the hospital. Chart I lists these deaths and gives the cause in greater detail.

The individuals operated upon in this series were nearly all quite elderly and many very feeble. The average age was 67.3 years, the youngest being thirty-five and the oldest eighty-nine. There were, however, five patients under forty years of age. It is interesting to note that three of these were patients in the Insane Asylum, the fracture occurring while under metrazol treatment. The fourth was injured in some kind of drunken brawl, the nature of which he cannot explain, and the fifth had many other fractures. Almost four out of five of our patients were females. The right hip was broken about as often as the left.

<center>RESULTS</center>

Anatomical Results. Fifty-nine cases have resulted in firm bony union proved by x-ray and rated by competent roentgenologists. Nine cases were rated as union forming on last x-rays available. Whether they have or will go on to firm union or not, we do not know as no further check has been made in this regard. Several are fairly recent and the others have been lost sight of. Twenty-six cases have been rated by us as clinically united, but in which no late x-ray is available. In this group, however, function is excellent or good in every instance. The reason the check-up x-ray has not been obtained is, in all instances, because the patient refused to come in for such a procedure, generally with such a remark, "My leg is all right. I don't see why I should have another x-ray."

Sixteen cases have resulted in frank nonunion, the details of which are brought out in Chart II. Two cases have been lost sight of and the net result is not known, and seven patients have been operated upon too recently to appraise.

Boiling down these figures, when one removes the deaths and cases lost from sight or too early to appraise, we have a total of 110 cases.

	No.	Per Cent
Firm bony union	59	53.63
Union forming when last x-rayed	9	8.18
Excellent clinical results without check-up x-ray	26	23.63
Nonunion	16	14.54
Total	110	

Inasmuch as it is not probable that any of those put down as clinically united are really ununited, in view of the function, it is probable that the percentage of nonunion is adequately stated. However, listing those only in which definite x-ray evidence is available, the following figures are obtained:

	No.	Per Cent
Known bony union	59	78.66
Known nonunion	16	21.33
Total	75	

These sixteen cases of nonunion, however, bring out some interesting facts when studied more clearly. Chart 11 goes into this in greater detail.

FUNCTIONAL RESULTS

The functional results are as follows:

	No.	Per Cent
Rated as excellent function	55	55
Rated as good function	24	24
Rated as fair function	12	12
Rated as poor function	9	9
Total	100	

There are several others which are not rated at all, either the patients died, the operations were done too recently or a mental condition or another disease has become superimposed upon the hip fracture, and the patient either cannot or will not even try to walk. Several such patients are known to have good bony union with perfect anatomical position. It might be assumed that the function would be excellent in these latter cases if the patients would or could walk, but we thought it better not to score these from a functional point at all.

It is interesting to note in this connection that some of our cases of known nonunion function well, whereas in some of our cases of known union, the function is only fair or even poor. One of these deserves particular comment:

This is Case No. 23, a woman forty-three years of age, who received a transverse central fracture of the neck of the femur in May, 1938. She was pinned on the day following the accident, was out of bed in five days and

home from the hospital in fourteen. For some fifteen months she did very well, walked without pain, limp or any loss of function whatsoever. However, in the late summer of 1939 she began to develop pain in the hip and went to a surgeon in another city who believed and told her that one of the pins was too long and should be removed. She submitted to this operation, having all three pins removed, but felt no better. Her leg was strong and there was no shortening, but there was considerable pain. She finally appealed to me almost four years after the original operation with what she thought to be several inches of shortening, great limp and considerable pain. The x-ray shows a dead head, which has worn off to such a degree that abduction is impossible, and although there is no real shortening in this leg, the apparent shortening, because of this adduction deformity, is about 3 inches. This case represents one of four known dead heads in this series. The other three ended in nonunion. This one, however, united.

There is another factor in these cases which is quite significant and that is the time element. The majority of patients were operated upon promptly. Three times the operation was done on the day of injury. All three of these patients did well. More frequently the operation was performed on the next or the following day. However, sometimes we have had to wait because of intercurrent conditions, in one instance as much as thirty days. This latter case also did well. In two cases which were done approximately sixty and seventy days, respectively, following the injury, nonunion occurred, one of which went on to union following a Schantz osteotomy. The length of time patients have remained in bed has usually been short. At least three patients have walked on their legs (without permission) on the day of operation, and all three of these went on to good union. Many patients have been out of bed within three or four days and unless some intercurrent condition was superimposed, the average stay in bed was less than a week. Many of our patients bore some weight within less than a week. It is true that in many of these patients, the neck shortened a little, but it is equally true that in some of these the neck did not shorten. It is also true that in many of those who remained in bed for months, the neck sometimes shortened or sometimes did not shorten. We believe that shortening of the neck is due to interference with the blood supply and not to early weight bearing; and that if it is going to occur (and one can never know this for certain), weight bearing on properly applied pins is probably an advantage.

CONCLUSIONS

We wish to present a method of internal fixation of fracture of the neck of the femur, which in our hands has been found satisfactory.

One must use good materials, and these materials must be introduced into a femur, the fracture of which has been accurately reduced, and so introduced that the fragments are held so firmly together that no other splinting is necessary. While our records of cases in which the result is accurately known show 78.66 per cent of

NONUNION

Case No	Age	Cause of Nonunion	Remarks
1	45	Gross infection	Has fibrous union with good function, about $3/4$ inch shortening, walks well
2	44	Dead head	Was first in a cast, complaining bitterly of pain Then pinned on tenth day after accident Continued to complain bitterly of pain Pins became loose in six weeks and patient was repinned Continued to complain bitterly of pain for several months Ended up with complete necrosis of the head, about $1\frac{1}{2}$ inches shortening, walks fairly well and tends to business as merchant
6	61	Dead head	Patient got along fairly well for several months and was walking Gradually began to have pain Pins loosened up and x-ray showed a dead head with upward slipping of the lower fragment Patient had good function Had pain for about one year which gradually subsided Returned four years later, died of malignancy of kidney, was walking well at that time
9	70	Failure of bones to grow	Pins became quite loose, patient already badly crippled with arthritis
12	51	Dead head	Patient complained bitterly of pain for about two months after pinning, walks well now but has considerable limp and considerable shortening
51	35	Insane patient	Insane patient, this was a metrazol case, patient already confined to insane asylum and still there, no co-operation
58	89	Failure of bones to grow	Patient was very feeble, walked on the pins for about a year Pins then became loose and fracture fell apart Was repinned but died a few months after this without ever gaining union, function for the first year very good
60		Insane patient	Metrazol case, had pneumonia, still confined to insane asylum
63	40	Rheumatoid arthritis	This confined her to bed for months before the fracture, complete nonunion, is still in bed but has no pain
77	75	Pinned too late following injury	Injury had occurred many weeks before, impossible to know how long, as the original injury was probably an impacted one and the second injury of at least one month before, breaking the impaction
87	67	Pinned too late following injury	Patient pinned at least ten weeks after injury, preceding which there had been no treatment, patient got union with Schantz osteotomy
91		Insane patient	No co operation
95	85	Improper introduction of pins	X-ray broke down at the time of operation and all pinning was done blindly and was not done well
105		Insane patient	No co-operation
112	80	No doubt due to multiple injuries following pinning	Patient became delirious, fell out of bed several times, had to be shackled, then jerked and pulled the shackles continuously, died in about three months of heart condition
113	83	Failure of bones to grow	Patient returned one month after pinning with thrombosis of popliteal and gangrene of leg, did not survive amputation but it was apparent from condition of hip that union would not occur

union, we believe that this figure is really entirely too low. There are several of our cases of nonunion in which union was not even expected; two were definitely done too late. Other cases have failed to unite because of conditions beyond our control or what in view of our present knowledge appears to have been an inefficient application of the method. We firmly believe that when one is confronted with a new case, properly selected, it can be assumed that in at least nine out of ten times union will occur.

DEATHS

Case No.	Age	Postoperative Day of Death	Cause
3	89	3	Cardiorenal
10	89	5	Uremia
19	86	40	Inanition
32	63	16	Coronary
40	79	6	Pneumonia
46	76	8	Coronary
59	73	14	Coronary
72	78	6	Pneumonia
120	72	6	Cerebral accident

DISCUSSION

N. J. Giannestras (Cincinnati, Ohio): Talking about fractured hips in Boston is like bringing coals to Newcastle. Some of these patients were interesting, particularly the ones we had at the insane asylum. The first individual I had was a "metrazol" fracture, and at the end of six weeks when we took our first check-up plate after the operative plates, I noticed that he had excellent union there and I happened to make that remark in his presence at the time, and he said, "Doctor, do you know how I got a good leg out of it?" I said, "No." He said, "From the second day after the operation, I jumped off the bed onto the floor so my leg would get good and strong." Perhaps you can imagine a man who weighed about 160 pounds jumping off the bed, about three feet high, onto a concrete floor and those pins holding. They seemed to do the work.

There is another interesting thing about this procedure, and that is the possibility of getting them up much earlier. Dr. Carothers had a case of a diaphragmatic hernia in which he was told that he would have to pin that

hip right away because if that woman did not get up, she would not be able to eliminate properly because of the diaphragmatic hernia, and that she would get in trouble. I believe that she was able to sit up and was up in about three or four hours after the operation.

Now, as Dr. Carothers mentioned, there is nothing new about pinning but it is simply the method of fixation. We have seen cases in which the neck has shortened but because the pins are parallel, they have either gone into the acetabulum for a slight distance, and then even though in some instances the pins have been impinging upon the acetabulum, it does not seem to make any particular difference and does not seem to cause any particular pain in that individual. In other instances, pain has been induced by impingement of the pins in the acetabulum and they have been removed.

Another observation is that, of late, they have found that it is sometimes a good idea to place the head in a little valgus position in comparison to the neck so that when the patients are up and around, they get a more definite thrust rather than a shearing stress at the point of fracture, and thus probably help in union.

E. PAYNE PALMER (Phoenix, Ariz.): This excellent paper and the splendid discussions leave very little to be said regarding the fracture itself. The medical profession is derelict in its duty when it does not explain to a patient who has a fracture of the neck of the femur that a large percentage of these patients are going to have crippling, that the percentage of satisfactory results has been increased by the various methods that are being used but we still have this percentage which is appalling, and as Dr. Speed says, this is still an unsolved problem.

So if this organization could do anything toward directing the profession as a whole to tell these people, when they get a fracture of the femur, and to tell the family, that there is going to be a percentage of failure which they have to anticipate, it would do a lot to unload the responsibilities of the man who treats fractures of the neck of the femur. Please remember this.

CAPTAIN BOARDMAN M. BOSWORTH (New York, N. Y.): I hesitate to impose upon you by taking the floor, and my only excuse is that in a way I represent another man. I am speaking for Dr. Preston Wad, a member of this organization, without his foreknowledge and consent, so I hope my remarks will not be taken too seriously.

There are just two or three questions that I wish to put to Dr. Carothers. The first is, do you use three pins all the time, or do you sometimes use four? I ask that particularly because we have had the sad experience in two cases of having the pins break within the first six months or a year, and in one of those cases the distal half of the pin we found located right in front of the bladder when we went to remove it. I wonder whether you have had the experience of pins breaking, and also whether you have had any of your pins wander out the other way, in other words, extrude themselves.

I also did not understand exactly how long your average follow-up was. At the City Hospital where Dr. Wade and I are on the staff, on Dr. Ban-

croft's staff, for the past five or six years we have had the privilege of handling all the fractured necks of the femur that came in on Dr. Bancroft's service, and as a matter of fact we are right in the middle of preparing a paper to give the follow-up results on 200 cases of fractured necks of the femur, both intracapsular and intratrochanteric, which we had nailed during that time. Unfortunately, that paper had to be postponed, but these questions arise as a result of our experience in that work.

I would like very much to have Dr. Carothers say whether he has a definite time at which he urges his patients not only to get out of bed, but to bear weight, first with crutches, and secondly at what time he likes to have them begin complete weight-bearing. Our policy has been what I presume his is, to get the patients out of bed and active as soon as possible. In other words, we have our patients, on the average, out of bed either the next day following operation, or the second day, with very few exceptions, and we have them up either on crutches or on a creeper within the first ten days to two weeks.

Then in the technic, I was very much interested in the angle iron with the holes in it that was shown. That struck me as a very clever arrangement. It had not occurred to us. We have been using two pennies, one of which we apply with adhesive over the major portion of Poupart's ligament. The other we place over the femur where we think we are going to put our pins in, and then we take our x-ray before operation, and those two penny marks are fixed with gentian violet or similar dye before the patient is prepared. We use those as guides very satisfactorily.

Finally, I may say that we, too, have had some experience with insane patients. Both of us have taken trips out to Kings Park Hospital on Long Island to nail hips which have been fractured by metrazol.

KENNETH M. LEWIS (New York, N. Y.): I would like to confirm an observation that Dr. Carothers made. At Bellevue we have been using Smith-Petersen nails. We have tried to get ambulatory Thomas splints for our patients to walk in, and we have always tried to avoid early weight-bearing, apropos of Smith-Petersen's dictum that when they do bear weight, there is a shearing force and a compression force between the fragments. Unfortunately, we cannot get ambulatory splints for all of them and they are put on crutches, and we have had a fair number of them—I should say fifteen or twenty of them—who simply would not use their crutches. They would be gotten out of bed and for the first few days would be in a wheel chair, but they were walking at the end of possibly a week or ten days, and I would come on the wards and see some of these people with their crutches over their shoulders, parading up and down the wards.

When the first few were observed, I was sure something was going to go wrong, but surprisingly enough, every one of those cases got union, and some of them got union very rapidly. This is the first observation I have ever heard made confirming the findings that we have had in a few cases. Why they get union any more rapidly than the others, I do not know. Ac-

cording to Smith-Petersen's idea of a shearing force, that should interfere with union, but in these cases of ours we have had the same experience that Dr. Carothers has had.

H. Gurth Pretty (Montreal, Canada): Dr. Carothers' paper has interested me very much, and particularly the fact that he has made reference to his follow-up cases in a period of five or six years. We have been reviewing some of ours in the last two or three months, and in the follow-up there are two points that I think are worth mentioning: First of all, in a group of about seventy-five cases, using a single pin, we have had four cases that have developed an adventitious bursa over the head of the nail, and that developed in a period of about eight months or a year; and by excising the bursa or extracting the nail at about eighteen months' time, our difficulties ceased.

On reviewing several of our cases which have gone a period of six years, we have been surprised to find that in three of them, they are developing a small triangular aseptic necrosis of the head at the apex of the nail. The union is perfect, the lines of stress and force are perfect, and in only one case did this individual complain of a suggestion of pain. The others had a slight click. But the interesting thing is that at the apex of the nail, in two cases in which the nail had been extracted, one in which it had not, there was this little triangular area of aseptic necrosis.

In checking up a group of old cases that had been treated by the Whitman method, or modifications of plaster, and looking over those, over a period of six and eight years we found that some of these had a similar condition but instead of occurring at the apex of the nail, it was occurring on the interior border of the neck, or rather the head.

I merely bring this up as an interesting point because we are interested to see what is going to happen and it may come to the point that we may have to do a reconstruction operation sometime in the future or put in some of the vitallium cups.

William Darrach (New York, N. Y.): I think it is hard for anybody to keep quiet on hips. I think it is still an unsettled problem. We are struggling to find an answer and we are still in doubt about a good many details. You think you get an idea along one line, and then you talk to somebody else and he brings in some more facts and things and it unsettles you.

I have been distinctly unsettled by the discussion this morning, and I am beginning to wonder about two points. I think it is quite outstanding that the author has had only one flat head in a series as large as that. We certainly can beat him on that because we have had a great many more flat heads develop during the second, third and fourth years, and because of that, I am wondering whether the three or four pins may not be a better bet than the Smith-Petersen pin which we have been using.

The other disturbing factor to me is the early weight-bearing. We have been fairly convinced that the percentage of flat heads, with or without bony union, is greater if the cases start weight-bearing early. I do not know

whether we are right or wrong, and as the figures are coming in from a good many places where early weight-bearing has been followed out, we are beginning to wobble in our position that at least six months should be allowed before any weight-bearing is tried. That, I do not know.

There is one thing I do know though, and that is we are delighted with the improvement in our results since we gave up using caliper splints in the upper treatment. The more I think about the caliper splint, the more ideal I think it is as one method of destroying any union that may be starting. The ring of the splint makes a beautiful fulcrum right over the site of fracture, not only for flexion, extension and abduction but rotation, and I think that our earlier experience using the caliper splint was perhaps one reason why our earlier results were not as good as they should be, and were not as good as they have been more recently since we gave up the caliper splint.

Of course, no matter how firmly you lay down the rule to these people that they must not bear weight, a great many of them do, so we are sneaking in a little series of early weight-bearings against our advice, but it is going to take another good many years to be able to tell what is the main reason for the late changes that come and disturb us and give painful hips, becoming more and more difficult and painful as the years go on.

I got another jounce from Dr. Gordon, and that is that perhaps the biochemist is going to help us. He spoke of these old ladies whose spines begin to decalcify and they get more and more kyphotic and compress two or three vertebrae, and he pointed out the similarity between those and the old ladies who break their hips, and the decalcification there and the later troubles in the heads may be in the same group. I think if they can help us in those cases of back injuries, they may help us on the hips.

Just to sum up, I have no firm convictions today, any more than I had thirteen years ago when I began to nail hips, but I think it is an interesting problem and I think we are learning something every year. I think Dr. Carothers has contributed a very real something with this series which he is reporting.

CHARLES S. VENABLE (San Antonio, Texas): There is one thought that I would like to inject into this discussion; that is, postoperative posture in avoiding adduction. The point of complete rest of the neck of the femur is with the inner condyle of the femur in a line with the superior spine. That is a neutral point. When those patients are permitted to drag the leg in adduction, you are putting a fulcrum right on the top of the fracture, and if they keep doing that, they may work the nail or the pins, or whatever you have, out. I have seen it occur. I have seen two cases in which I had to put a nail in, but the patient would not maintain that reasonable position, so I put a simple hip spike on. I am permitting them to be ambulatory with crutches, with minimum weight-bearing, driving it back because in adduction they do that. I have seen two cases of bent screws or nails just from that.

RALPH G. CAROTHERS (closing): I want to thank the gentlemen very much, and I hope I will answer all the questions.

In regard to early weight-bearing, let me repeat, of all those cases that did well, 80 per cent bore weight early, within a week or ten days. Of those who did badly, only 20 per cent bore weight within that time.

We have attempted in every case to get a little valgus deformity in our reduction. We have not always succeeded but we have tried to do it. We have tried in every case to get the pins very low, and we hope that in these instances we have so pinned the hip that there is not a shearing force but a pressure force; and for those reasons, I believe the neck will shorten sometimes, no matter whether they bear weight or not.

I have one case of a greenstick fracture of the forearm of a child, in which the check x-ray, when we finished with it, showed an absolutely hair-line fracture with perfect position. It was put in a plaster cast and six weeks later when the cast was taken off, there was several millimeters' distance between the ends of the bone. The ends of those two fragments had sloughed and there was no weight-bearing, and it was in good firm platers all that time.

I have seen the same thing when transverse fractures have been treated with plates in which the ends of the bone slough and the plate holds the fragments apart, thereby preventing the collapse of the fragments but also preventing union. But in this sort of thing it will not happen.

I cannot agree with Dr. Speed—and I know I am going out on an awful limb to disagree with him—about this early weight-bearing, but we learn from some crazy people. Some of our bad luck came in the insane, but some of our very best luck came with those who bore weight early; and one of the very early good cases that we had was walking right down the hall that same night she was operated upon. That has happened on several occasions and the bones have united, so we began getting them up after that, and getting them up early, and we have had far better luck since we did that. Our bad results are mostly in the beginning.

Now, the biochemist is going to come into this thing some way or other, and I do not know just how, but the first good case I had, and the first good case in this whole series, was the elderly lady that Dr. Giannestras spoke about who had the diaphgramatic hernia and also had pernicious anemia and diabetes. She was getting liver, and I think was getting hydrochloric acid and insulin. Whether they had anything to do with it or not, I do not know, but that old lady lived for four years and I know her hip was good at the end of four years because I got a check picture on it and I talked to her about it.

We have had some pins come out. Some have been loosened up and have extruded. They have been mostly those pins that were put in in too transverse a fashion, too high up, and it is in that converging pin situation where you do get a wiggling like that, but when you have it sliding on it nothing

happens. If you get it up that way, then it does loosen. None have gone into the pelvis, none have broken. And please do not call them wires. That is not a wire. You cannot buy this pin. You have to make your own to make a good one. It is an 18.8 stainless steel that is properly rolled and you cannot break it. Since we got them rolled right, none of them have even bent.

The follow-up has varied from seven years to a few months. Those that have been done within three months have been put down as too early. The weight-bearing has been anywhere from a few hours to never, but in the good cases, it has almost always come within a week or ten days. The average case is gotten out of bed the next day and is bearing weight with a walker, crutches or some sort of thing within a week to ten days. Of course, some people cannot get out of bed. Half a dozen of our cases had strokes when they fell and were paralyzed. Some fell with an attack of angina and their physician made us keep them in bed, and so we could not get them up.

We have used no type of external splinting of any sort whatsoever. We have in our intertrochanterics. That is another story. We have used something to keep them from rolling out.

I do not know why we have had less head flattening. Maybe we will get some more, but we just have not had it; and I do believe that these pins do not chew up the head quite as much as a Smith-Petersen nail does.

ROENTGENOLOGICAL MANIFESTATIONS OF BONE REPAIR

HEALING OF FRACTURES WITHOUT EXTERNAL CALLUS

ROBERT G. VANCE, M.D. AND CAPT. GEORGE M. WYATT, M.D.

Diplomate of the American Board of Radiology; Assistant Roentgenologist, Massachusetts General Hospital

Diplomate of the American Board of Radiology; Formerly Assistant Roentgenologist, Massachusetts General Hospital

BOSTON, MASSACHUSETTS

THE physiological process of bone repair following fracture is a complicated one.[2] It may be summarized briefly as: (1) Hemorrhage in and about the fracture; (2) decalcification and resorption of devitalized bone; (3) ingrowth of granulation tissue about the fracture; (4) formation of osseous and, to a variable degree, cartilaginous matrix; (5) calcification and organization of this matrix to form bony union, and (6) shaping of the new bone to its final contour.

Roentgenological examination can accurately demonstrate only part of this process,—namely, alterations in the amount and distribution of calcium in the region of the fracture. The other phases of bone repair can, however, be largely inferred by the roentgenological appearance with the understanding that the changes visible on the films are gross and lag considerably behind the histological process. If serial roentgenological examinations are made, keeping in mind the above physiological processes, a decrease in the density of the fracture margins will be seen at about the tenth day. This, we believe, represents the decalcification and resorption of devitalized bone which is a preliminary step in bone repair. After two or three weeks, or sometimes earlier in children, we may see calcification in the matrix surrounding the margins of the fracture which is termed callus. This is the fusiform calcification which we are eager to see and which reassures us that healing is progressing normally. This type of callus is termed external callus.

At the same time a similar process is taking place between the margins of the fracture, uniting the contiguous surfaces. This callus is known as internal or intermediate callus and is not as clearly visible in the roentgenogram as external callus due to the density of the adjacent bone and absence of a soft tissue background. External

callus, on the other hand, is thrown into relief by the surrounding radiolucent soft tissues and can be seen earlier and more clearly than internal callus for this reason.

The roentgenological visualization of external callus is important for the following reasons: (1) Its presence reassures the physician and the patient that healing is taking place. After checking the position of the fragments, the next question of the physician is, Does the x-ray show any callus? By this he usually means external callus. (2) The appearance of callus gives some indication of when it is safe to remove splints or other apparatus. (3) It offers a guide as to when the injured part may resume function. (4) The presence and character of external callus is sometimes used to determine the lapse of time since injury. This is frequently important in medicolegal work.

The appearance of external callus is so well known that its further description here is not warranted. Its presence may be comforting, but its absence does not necessarily mean that union will not take place.

An occasional fracture in any location may heal without visible external callus though this is exceptional in the shafts of long bones. The purpose of this paper is to call to attention a certain group of fractures which heal promptly, but do not show external callus formation as a part of the healing process.

When fractures are healing without external callus, it is important to recognize such healing for the following reasons: (1) If it is inferred that union is not taking place simply because no external callus is visible roentgenologically, the physician and patient may become unduly discouraged. (2) If union without external callus is not recognized, splints or other immobilizing apparatus may be employed over an unnecessarily long period of time, thereby increasing atrophy of bone and soft tissue. (3) Medicolegal testimony in which the age of a fracture is estimated by the absence of external callus only, may be fallacious.

One of us (R. G. V.) has previously described the repair of linear fractures of the bones of the cranial vault in which healing occurs without visible external callus.[4]

A recent extensive roentgenological study of phalangeal fractures showed no callus formation about fractures of the terminal tufts although these fractures practically always united.[3] These fractures are very common in industry and they may be clinically well in three weeks but show no union roentgenologically for several months. Thus many days of labor may be lost if the patient is not returned to

work before there is roentgenological evidence of union. This is very important in these days of labor shortage.

It is well known that fractures of the vertebral bodies heal without roentgenological evidence of external callus.

Observation of a large number of fractures led us to believe that fractures involving joints or within joint capsules were also prone to heal without the formation of external callus visible in the roentgenogram. Accordingly, a review of six hundred cases of fracture was made in an attempt to ascertain the presence or absence of external callus in such fractures. Of these six hundred cases, seventy-two were selected for critical study according to the following criteria: (1) Only fractures which lay within or extended to a point within a joint capsule were included. (2) Only cases with examinations at proper intervals to demonstrate callus and with films of a quality that would show a small amount of callus were included.

Obviously a large number of cases were excluded because of heavy casts or other apparatus which obscured the region of the fracture, because a portable machine was used, or as in the case of fractures of the lateral malleoli, only a follow-up examination was made to determine position.

The films of the seventy-two selected cases were studied with particular attention to callus formation. The following points were noted in this study: (1) No external callus was seen at any time about any fracture margin which was within a joint capsule. (2) No external callus was seen in fractures of the tuberosity of the humerus where the tuberosity alone was involved or in fractures of the malleoli below the level of the tibio-astragalar ligament. Frequently, no external callus was seen in the above locations even when the fracture line extended considerably beyond the joint capsule.

In those patients in whom healing took place without external callus the first roentgenological sign of healing was an increase in the density of the fracture line accompanied by a decrease in the sharpness of definition of the fracture margins. This change could be detected after an average period of four weeks which is later than the average appearance of visible external callus.

Further healing, following the initial increase in density consisted of a gradual obliteration of the fracture line with formation of new trabeculas to join those of the adjacent fragments. There is marked variation in the time required for this process ranging from eight weeks to eight months. The degree of separation of the fragments plays a very important rôle; the greater the separation the larger the time necessary for complete healing.

No conclusive explanation is available for the lack of external callus within joint capsules although an inhibitory action has been attributed to synovial fluid.[1] The lack of an explanation does not, however, alter the fact that external callus is inhibited in these locations. In the clinical care of patients with fractures involving joints, it is important to recognize that such fractures can heal without external callus being visible roentgenologically at any time in the course of repair.

CONCLUSIONS

1. Fractures in all locations may heal firmly and promptly without showing external callus visible by roentgenogram.

2. Healing without visible external callus is the rule in those fractures which lie within joint capsules.

3. Healing without visible external callus frequently takes place in fractures which extend into joint capsules even though part of the fracture lies outside of the capsule.

4. It is fallacious to assume that a fracture is of recent origin because visible external callus only is absent.

REFERENCES

1. COTTON, F. J. Dislocations and Joint Fractures. 2nd ed., p. 36. Philadelphia, 1924. W. B. Saunders Company.

2. SCUDDER, C. L. The Treatment of Fractures. 11th ed., pp. 158–207. Philadelphia, 1938. W. B. Saunders Company.

3. SMITH, F. L. and RIDER, D. L. A study of the healing of one hundred consecutive phalangeal fractures. *J. Bone & Joint Surg.*, 17: 91–109, 1935.

4. VANCE, R. G. Healing of linear fractures of the skull. *Am. J. Roentgenol.*, 36: 744–747, 1936.

DISCUSSION

PHILIP H. KREUSCHER (Chicago, Ill.): The paper by Dr. Vance was interesting to me because of the fact that I have been in error on many occasions, I suppose most of us have, in saying that some of these patients were actually not healed, and we have been in error in not permitting the patients to go back to work or to go back to their usual job in the time when we really should have known that they were healed.

The medicolegal aspects come into the picture. It is difficult to convince a jury, and it is a very difficult thing to convince a Commissioner in an Industrial Commission, an Arbitrator, that a fracture is healed when there is no definite external callus. They have been taught, or at least they think they know, that there must be an external callus before the thing can be healed.

I should like to ask Dr. Vance if he has had any experience with a refracture in some of these cases. In other words, does a refracture take

place more commonly or with less provocation than where there is actually a good deal of external callus present?

Congenital defects, I believe, must be considered in all of these cases, but I should like to make one point and that is, when a patient has one congenital defect, such as a clubfoot or hairlip, or some other congenital defect, look out for some defect also in his spine or some other part of his skeleton. When you have a patient who has a spina bifida occulta or has some other congenital defect in the spine, look out for a congenital malformation of one of the vertebrae also, even an absence of one of the vertebrae.

CHARLES S. VENABLE (San Antonio, Texas): In speaking of Dr. Vance's paper and the question of callus in fractures about the joints, I rather think that it is important for us to recognize that we should not expect callus about spongy bone as we do in diaphyseal bone. We may find a line of union as it goes by the progressive deposit of lime salts in the fracture site, but in the thinner cortex of harder bone about the spongy bone, the process is a different one and it is a much slower process of union.

As you go further into the shaft from a fracture, you see the callus about the fracture in the lower third of the fibula, while you do not see any callus at the malleolus in a femur. I think that is true in fractures of the neck of the femur. We hear it said, "I do not see any callus." I do not think you ever see any callus about the neck of the femur unless it is in malposition. If it is in malposition, you begin to have a pile-up of malleable bone which makes for callus. But in proper position, I think you only see it through the line of fracture in deposit. I think Dr. Vance has done an excellent job in emphasizing that to us in our reading of x-rays.

HENRY C. MARBLE (Boston, Mass.): Before you close the discussion, Dr. Vance, I want to ask one question. Dr. Darrach and Dr. Murray in New York have us plating tibias and femurs so perfectly that after they have been plated, the patient gets well and walks around and we never see any callus. Does that fall into the same category?

ROBERT G. VANCE (closing): When I showed you that humerus, I called attention to the fact that any bone anywhere may heal that way. I do not know why.

I wish to thank Dr. Kreuscher and Dr. Venable for their discussion of my paper. In reply to Dr. Kreuscher's question about refracture, we have not seen refractures commonly in that type of fracture in which healing has occurred by internal callus.

SOME OF THE USES OF THE CUTIS GRAFT IN SURGERY*

JOHN E. CANNADAY, M.D.

CHARLESTON, WEST VIRGINIA

IN December, 1941, I read a paper[1] at the Pinehurst meeting of the Southern Surgical Association on some of the uses of the cutis graft in surgery and reported a series of illustrative case reports. Since that time I have had further experience with this method and am making an additional report.

This method was apparently first used by Loewe,[2] later by Rehn,[3] of Germany, and later still by Eitner,[4] of Austria, in plastic and reconstructive surgery. It has apparently been used more generally and frequently in Germany than elsewhere. Rather extensive use has been made of the cutis graft by many of the leading plastic surgeons of this country. Among them are Blair,[5] Brown,[6] Byars,[7] also Davis,[8] Ivy,[9] Fomon,[10] Bames,[11] Straatsma,[12] Smith,[13] Peer[14] and Paddock. Uihlein,[15] after checking over 104 cutis graft cases that underwent operation by Rehn at the University Clinic of Freiburg, was able to trace eighty-four patients. The late results in seventy were good; in fourteen, poor. While this method has been used to a considerable extent in Europe it has not received much attention from general surgeons in the United States.

Rehn[16] and his co-workers, from their experience in animal experimentation, and with the clinical use of the dermal graft, arrived at the conclusion that cutis graft tissue takes on the function and characteristics of the tissue that it replaces; to be more specific, that it may be converted into tissue resembling tendon or that it may assume the characteristics of the various muscular aponeuroses of the abdominal wall.

The usual fate of the cutis graft that has healed in the body tissues is that of a slow conversion into fibrous tissue. Peer[17] and Paddock, in the course of a large series of nasal reconstruction operations, stored pieces of derma along with cartilage beneath the skin for varying lengths of time, from two weeks up to two years. Histological sections were made from these and studied. They found that a gradual process of degeneration of the normal skin structures took

place, that the sweat glands and the hair follicles were the first to disappear and that a gradual infiltration and replacement by fibrous tissue took place.

FIG 1. Large incisional hernia previous to operation (From *Ann. Surg.*, vol 115, no 5, May, 1942)

FIG 2 Hernia ten weeks after operation (From *Ann Surg*, vol 115, no 5, May, 1942)

Cutis graft material can be readily prepared by anyone who is accustomed to the use of the skin graft knife. It has the merit of being easily obtainable at all times and of being more readily available than fascia. It is superior to fascia in many respects. Some have suggested that its use might lead to the formation of epidermoid cysts but the observations by various European surgeons over long periods of time, likewise those of various plastic surgeons in this country, have shown that with proper technic such possibilities are apparently too remote for very serious consideration. It has been definitely proved that the cutis graft gives strong support from the beginning of its placement, that it heals in promptly and that it is gradually converted into fibrous tissue and remains as a definitely firm support wherever it has been placed.

OPERATIVE TECHNIC

The operative procedure for the use of the cutis graft is essentially similar to that used for implantation of fascia. After the defect has

been surgically exposed and repaired as well as possible by suture methods, a graft of suitable shape and size is taken from the antero-external surface of the upper third of the thigh, (although the skin

FIG. 3. Large inguinal hernia.

from other areas may be almost as suitable), the epidermal layer being cut away in a thin sheet with a skin graft razor, after which an area of derma of suitable shape and size is outlined and removed from its bed. A thin layer of subcutaneous fat may or may not be left on the undersurface of the graft. This particular detail apparently makes but little difference. The graft is fixed in place in firm tension with interrupted sutures usually of No. 40 cotton thread. A moderate amount of sulfanilamide, also sulfathiazole, is routinely placed underneath as well as above the graft. Usually not more than 2 to 3 Gm. of each is used in the average incisional hernia repair. Great care is taken to suture the subcutaneous structures in such a manner as to obliterate as nearly as possible all dead space in the region over the graft. No. 70 or No. 80 cotton thread is generally used for this purpose. In many cases, and especially when the graft is large, a bite is taken by the needle about the center of the graft by each individual suture crossing that area.

While the cutis graft transplant is often used as a reinforcement for defective structures, the graft may be satisfactorily used to bridge serious and extensive defects. Where unusual strength is required, a
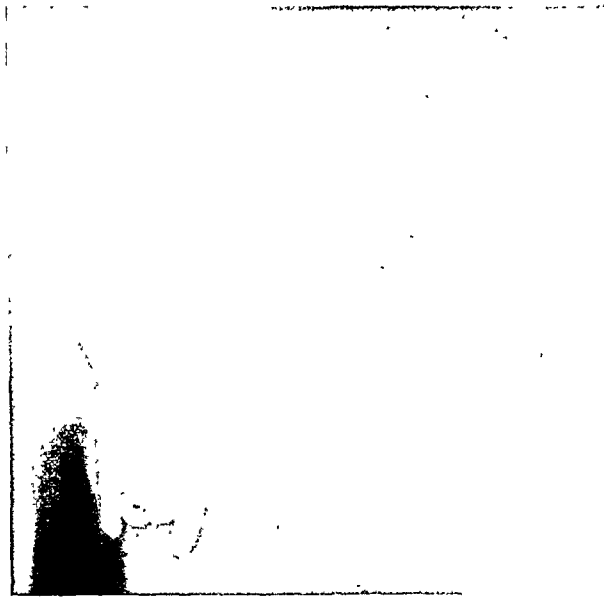
FIG. 4. Large lower abdominal incisional hernia previous to operation.

double thickness graft can be used with gratifying results. Through-and-through tension sutures are not used in these wound closures. Skin clips are applied, also fine superficial intervening sutures are used to approximate the skin edges. The suturing throughout the wound, likewise ligation of bleeding points, is carried out with due regard to principles laid down by Kocher[18] and later emphasized by Halsted[19] with reference to the use of sutures and ligatures.

When making use of the graft in connection with the repair of inguinal hernias we usually suture the deeper structures to Cooper's ligament, etc. The graft is then sewn in place and covered by the aponeurosis of the external oblique.

Although Rehn[20] advised keeping large hernial repair cases in bed for from six to ten weeks subsequent to operation, we have found this procedure to be quite unnecessary and usually encourage our patients, regardless of the size of the hernial opening, to be out of bed at least twice each day subsequent to the day of operation without untoward results.

The indications for the use of cutis transplants are essentially those for which fascia has been formerly so universally used. More

especially is it indicated for the repair of large hernias of any type in which the openings are so large or the adjacent structures are so weak or defective that a satisfactory repair cannot be made by the usual
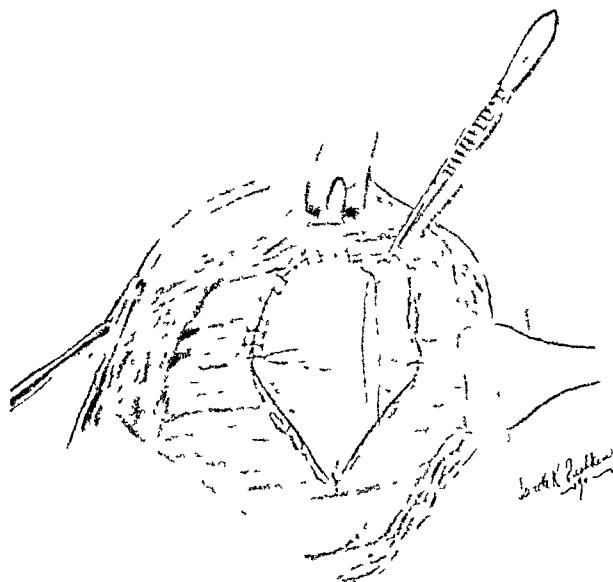
FIG. 5. Cutis graft being sutured to rectus aponeurosis.
(From *Ann. Surg.*, vol. 115, no. 5, May, 1942.)

overlapping methods, joint lining material in arthroplasties, the replacement of damaged dura, the filling out of defects in various parts of the body, more particularly about the head and face, as strong permanent autoplastic suture material wherever it may be needed, the replacement of damaged tendon, the suspension of the uterine cervix in cases of marked uterine prolapse, the reconstruction or reinforcement of weak or absent ligaments, the approximation of bone fragments in the treatment of fractures, either total or stage ligation of large blood vessels.

My surgical associates and I in the Charleston General Hospital have made use of cutis grafts in a total of thirty-seven cases, as follows: Three recurrent epigastric hernias (in one of these a small hematoma was evacuated a few days after operation); seven large incisional hernias of complicated types, not considered satisfactorily operable by the usual methods; seventeen direct and indirect inguinal hernias, which were either recurrent or in which the structures were so defective that satisfactory results could not be expected

by the usual types of repair, (of these last seven were operated by Dr. Bankhead Banks; one by Dr. Hugh Bailey; two by Dr. Victor Skaff, and seven by myself), all with apparent good results. In two
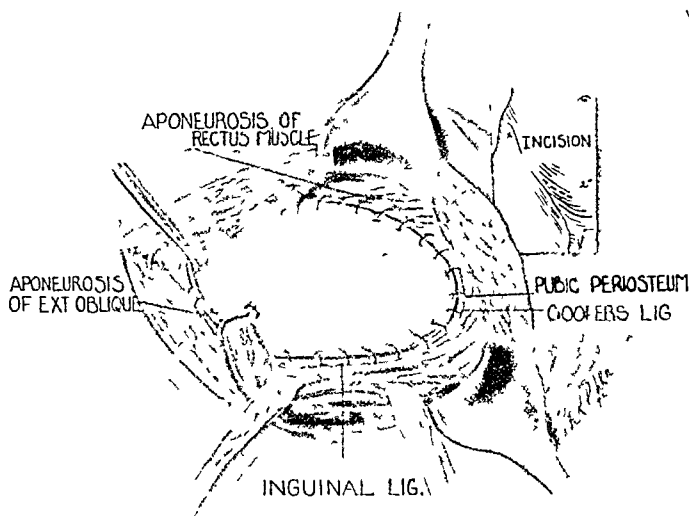
FIG 6. Cutis graft reinforcement for inguinal hernia with defective structures. (From *Ann. Surg.*, vol. 115, no. 5, May, 1942.)

cases cutis graft material was used to fix the uterine cervix to the undersurface of the rectus aponeurosis just above the pubis for the relief of uterine prolapse; in one case to secure in position an osteo-periosteal graft for non-union of an ulnar fracture; in one case for partial ligation of the femoral artery; in one case of double fracture of the patella; one case for replacement of torn dura; one case of abnormal mobility of the knee associated with defective right lateral ligaments, to support the bowel in two cases of sigmoid colostomy; one case of luxation of the acromial end of the clavicle.

In two of the cases enumerated there were mild infections. In one case a small superficial serum pocket developed but healed in about a week. In another case a moderately sized serum pocket developed in the subcutaneous fat. This persisted until the pocket was laid open after which it rapidly filled in by granulation. In none of the afore-mentioned cases was there any interference with a satisfactory end result.

CASE REPORTS

CASE 1.   B. P., a white male, age fifty-one, was admitted to the hospital May 27, 1940, with a very large epigastric hernia. The bulge on deep expira-

tion was fully 4½ inches across. He had a small ulcerated area over the hernia, apparently due to friction from his clothing. A wire screen was placed over this and the patient was kept in the hospital for three weeks at the end of which time healing had taken place.
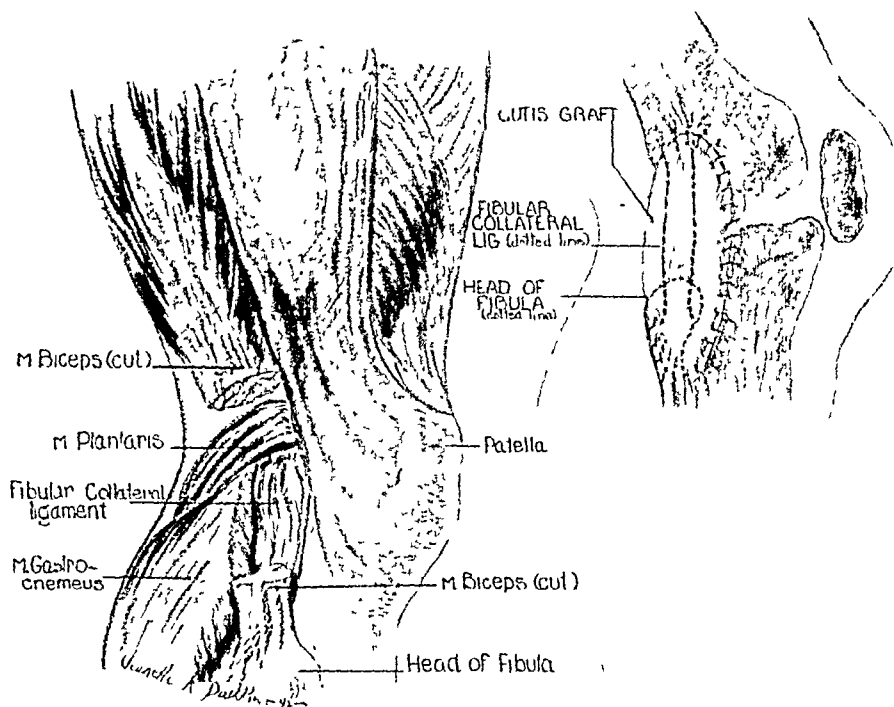
FIG. 7. Anatomy and cutis graft repair for unstable knee due to defective external lateral ligaments.

On July 9, 1940, the hernial sac was excised; the parietal peritoneum was closed by overlapping. The edges of the muscular aponeurosis could not be approximated. A cutis graft from the thigh was sutured over the defect. The patient had an uneventful convalescence. A recent check shows no evidence of recurrence. He returned to his former occupation of coal loading several months ago.

CASE 11. T. L. K., a white male, age sixty-nine was admittted to the hospital April 19, 1941, for repair of a large lower right incisional abdominal hernia. He weighed 170 pounds; his blood pressure was 170 systolic, 110 diastolic. The size of the hernial bulge was approximately that of an average baby's head.

At operation on May 8, 1941, the hernial sac was found to contain several loops of small bowel. The opening in the aponeurosis was approximately 4 by 2 inches. This was closed by use of a cutis graft from the

patient's thigh. Up to the time of his death from coronary sclerosis in May, 1942, no sign of a recurrence had been noted.

CASE III.   J. H. E., a white male, age fifty-eight, was admitted to the hospital December 30, 1941, with a very extensive incisional hernia in the right upper quadrant, following a gallbladder operation two years previously. There were five separate pockets in the hernial area.

At operation on January 8, 1942, two cutis grafts were used. The first was taken from the redundant tissue in one of the wound flaps and was sutured to the posterior fascia and parietal peritoneum. The remains of the fascia were sutured over this graft. A second cutis graft, from the right thigh, was sutured to the aponeurosis surrounding the hernial opening. The patient was discharged January 16, 1942. To date there have been no signs of recurrence.

CASE IV.   H. M., a white woman, age forty-two, height 5 feet 2 inches, weight 284 pounds, with a blood pressure of 220 systolic and 120 diastolic, had a large lower abdominal incisional hernia following cesarean section done elsewhere several years previously.

Operation was performed on May 6, 1942. There was an enormous hernial sac involving mainly the right side of the lower quadrant. When the patient was standing this sac extended within 3 or 4 inches of the knee. After mobilizing the hernial sac and excising most of it, the hernial opening in the abdominal wall was found to be 5½ inches across. By plicating the remains of the hernial sac and by utilizing interrupted sutures it was possible to bring the edges of the aponeurosis in approximation but not to overlap them. The suture line was then reinforced by a running cutis whipstitch for the entire length of the opening. This strip of cutis was taken from the edges of the operative incision.

She was discharged from the hospital May 27, 1942, at which time the incision was apparently well healed. A recent inquiry indicates that this patient remains in good condition.

CASE V.   R. B., a white male, age twenty, was admitted to the hospital July 13, 1942, with a compound depressed fracture of the left parietal region. His blood pressure was 130 systolic and 90 diastolic. There was a deep scalp laceration of the left parietal region. X-ray examination indicated that a portion of the depressed bone had been driven into the brain for the depth of about 1 inch.

Operation was performed on July 13, 1942, by the surgical resident, Dr. Victor Skaff. The traumatic incision was débrided and elongated. The depressed areas of bone were removed. A considerable amount of brain tissue escaped. The lacerated meningeal artery bled freely and was controlled by electrocoagulation. The area from which the bone was removed was about 1¾ inches in diameter. Since a considerable portion of the dura had been so badly lacerated that it could not be satisfactorily closed, a cutis graft was taken from the left thigh and sutured over the damaged

area of the dura. When in place the graft covered most of the opening in the skull. The temporalis muscle was sutured over the graft and the scalp was closed with interrupted sutures. The patient had an uneventful convalescence and was discharged from the hospital July 24, 1942, apparently in excellent general condition. When seen about six weeks later the area over the bony defect that had been covered by the graft was quite firm to the touch. It was apparent that the cutis graft had in considerable measure formed a protective covering over the area of brain that had been exposed.

CASE VI. L. S., a white woman, age fifty-eight, was admitted to the hospital August 3, 1942. She had a large, lower abdominal incisional hernia that had followed an abdominal operation performed about fifteen years previously. Her blood pressure was 180 systolic and 110 diastolic.

At operation on August 6, 1942, the patient's hernia was approximately 12 inches across. It was found that there were four distinct hernial openings rather closely linked together. The size of the wound after the different sacs had been mobilized was 10½ inches in its greatest diameter. The opening was partially closed by plicating sutures after which a large cutis graft was taken from the redundant skin over the hernial mass on the left side of the abdominal wall and sutured in place. This graft, when sutured in position, covered all four openings and extended about ¼ inch over the aponeurotic edges. The patient was discharged from the hospital August 27, 1942, with the incision well healed. A recent check has not shown any signs of recurrence of the hernia.

CASE VII. W. B. P., a white male, age eighty-four, was admitted to the hospital August 31, 1942. Examination showed a huge dissecting aneurysm of the left thigh. This had developed subsequent to the spontaneous subcutaneous rupture of a popliteal aneurysm of long standing. His thigh was tremendously swollen and the skin over most of it was ecchymotic. A terrific pulsating bruit was present.

At operation on August 9, 1942, a partial closure of the femoral artery with a strip of cutis carried twice around it and anchored with interrupted cotton sutures was made. Pulsation somewhat similar to that of a normal artery of the same size was elicited in the popliteal space for several days subsequent to the partial closure of the lumen of the artery. A slowly progressive dry gangrene, beginning in the great toe gradually ascended the leg. Amputation was suggested but declined. The patient died on August 30, 1942. Autopsy was refused.

CASE VIII. M. C., a white woman, age sixty-three, height 5 feet 6 inches, weighed 175 pounds. Her blood pressure was 170 systolic and 100 diastolic. An enormous right inguinal hernia was present; the sac extended down over the vulva, bordered the right labium minus and nearly reached the knee.

Operation on September 12, 1942, was performed by Dr. R. E. Pence and myself. The hernial sac contained numerous loops of small intestine. The sac, when mobilized, was 12 inches long by 5 inches wide, the dependent portion being the wider. The hernial opening was sufficiently large to admit the male hand (glove size 8½) and arm for general exploration of the abdominal cavity. The hernia was in part of the sliding type, the edge of the bladder peritoneum coming out onto the sac wall. The hernia was repaired as far as possible in the usual manner by suturing. A cutis graft was sutured over the repair. The aponeurosis of the external oblique was sutured over this.

This patient had an apparently normal convalescence. However, on the eighteenth day an abscess became apparent in the more dependent portion of the operative wound. This was opened after which prompt healing took place. So far there is no suggestion of recurrence of the hernia.

CASE IX. M. D., a white male, age forty-three, was admitted to the hospital September 22, 1942, with a history of an injury to the right knee received in a mine accident, which resulted in abnormal mobility of the right knee apparently associated with defective right lateral ligaments.

This patient was operated upon jointly on September 25, 1942, by Dr. R. L. Anderson and myself. A longitudinal incision was made on the outer side of the knee so as to expose the head of the fibula, also the external femoral condyle. A strip of cutis of suitable length and width was applied over both of these structures and secured in place with interrupted sutures. Healing was satisfactory but sufficient time has not yet elapsed to determine whether or not the result will be completely satisfactory.

CASE X. S. D., a colored male, age twenty-seven, was admitted to the hospital October 30, 1942, with a history of an injury to the left shoulder received in a mine accident, which resulted in inability to move the shoulder in a satisfactory manner. A diagnosis of dislocation of the acromial end of the left clavicle was made.

Under ether anesthesia operation was performed November 11, 1942, by Dr. H. A. Swart and myself. The Bunnell technic was used, a strip of cutis being substituted for fascia lata. Although this patient was apparently in vigorous health, his heart stopped beating and he died in the operating room just after completion of the operation. Autopsy was not obtained.

CONCLUSIONS

Cutis may be used in all cases in which the use of fascia or tendon might be indicated with the expectation of superior results. It heals in rapidly and well; is strong and stable from the time of operation; has great vitality; is able to survive under adverse conditions; possesses great tensile strength; has a good blood supply; gradually assumes the function of the part it replaces; is gradually converted into fibrous tissue and is readily available when needed.

REFERENCES

1. CANNADAY, JOHN E. *Ann. Surg.*, 115: 775, 1942.
2. LOEWE, O. *München. med. Wchnschr.*, 24: 1320, 1913; *München. med. Wchnschr.*, 51: 2125–2128, 1929.
3. REHN, E. *München. med. Wchnschr.*, 1: 118, 1914; REHN, E. and MIYAUCHI. *Arch. f. klin. Chir.*, 105: 1, 1914.
4. EITNER, ERNST. *Med. Klin.*, 4: 93–95, 1920.
5. BLAIR, VILRAY P. Personal communication.
6. BROWN, JAMES B. Personal communication.
7. BYARS, LOUIS T. Personal communication.
8. DAVIS, JOHN STAIGE. Personal communication.
9. IVY, ROBERT H. Manual of Standard Practice of Plastic and Maxillofacial Surgery. Philadelphia, 1942. W. B. Saunders Co.
10. FOMON, SAMUEL. The Surgery of Injury and Plastic Repair. P. 158. Baltimore, 1939. Williams & Wilkins.
11. BAMES, H. O. Personal communication.
12. STRAATSMA, C. R. Cited by Peer.[14]
13. SMITH, FERRIS. Manual of Standard Practice of Plastic and Maxillofacial Surgery. Philadelphia, 1942. W. B. Saunders.
14. PEER, LYNDON A. *J. A. M. A.*, 115: 357–360, 1940; PEER, LYNDON A. and PADDOCK, ROYCE. *Arch. Surg.*, 34: 268–290, 1937.
15. UIHLEN, ALFRED, JR. *Arch. Surg.*, 28: 118, 1939.
16. REHN, E. In Lexer, E.: Neue Deutsche Chir., Vol. 26 b, Die freien Transplantationen. Stuttgart, 1924. Ferd. Enke.
17. PEER, LYNDON A. and PADDOCK, ROYCE.[14]
18. KOCHER. Eine einfache Methode zur Erzielung sicherer Asepsis. *Corr.-Bl. f. schweiz. Aerzte*, 18: 3–20, 1888.
19. HALSTED, WILLIAM S. *J. A. M. A.*, 60: 1119–1126, 1913.
20. REHN, E. Cited by Uihlein.[16]

TRAUMA AND MALIGNANCY

WILLIAM J. CARSON, M.D.

Surgeon, St. Luke's Hospital

MILWAUKEE, WISCONSIN

IN the oldest known record, Imhotep[1] describes in detail forty-eight surgical cases, forty-five of which are due to injury. In Case 39, tumor in the breast, perhaps resulting from injury and in Case 45, benign tumor of the breast, he specifically states it is not due to injury as in Case 39. Since this was written 5,000 years ago, thousands of papers have been published for and against the concept of trauma causing malignancy. Cancer is mentioned in the papyrus Ebers (B.C. 1500). Hippocrates, Celsus and Galen described cancer. Leonides of Alesandria (A.D. 180) recommended radical excision, cutting through healthy tissue with knife and cautery. Lanfranchi of Lyons, in 1290, urged radical operation. Guy de Chaulliac (1300–1386) excised cancer with the knife. Paracelsus (1492–1541) believed that cancer was due to mineral salts in the blood, during which time there was a definite decline in the radical operation for cancer. Bichat,[2] in 1801, distinguished between the stroma as cellular and conceived of its growth by proliferation. Raspail[3] with the microscope, in 1826, showed that the growth of tissue resulted from the multiplication of cells. Schwann,[4] in 1838, established the doctrine of cellular structure as universal principle and discovered the nucleus and nucleolus of the cell. Muller[5] in the same year, published his study of malignant tumors, finding them all to contain nucleus and nucleolus. Virchow,[6] "while admitting the fact that the influence of traumas could not be dogmatically denied, stated that there must be some as yet unknown predisposing factor in the patient and also that the trauma must have been implanted on pathological tissues." Coley,[7] in 1898, reported 170 cases of sarcoma, forty-six of which gave a history of trauma. Ribbert[8] stated that a single trauma may lead to the production of a cancerous growth, Van Bergmann[9] that a single contusion could serve as a predisposing factor in mammary cancer. Ziegler,[10] in 1903, held that 7 to 14 per cent of malignant growths were of traumatic origin. Lowenstein[11] found 241 malignant tumors in soldiers, thirty-nine or 16.5 per cent due to trauma, one malign tumor to 15,000 injuries. Lewy[12] reported thirty-seven tumors in 26,389 injuries from the New York Industrial Commission. Mock

and Ellis,[13] in 1926, reported nine cases which they believed fulfilled the postulates. Villata[14] found 8 per cent of sarcomas and 5 per cent of carcinomas gave a history of single trauma. Mason,[15] in 1929, reported seven cases of carcinoma of the hand and collected twenty cases from the literature due to trauma. Dietrich[16] states if we should regard tumors as arising independent of an anlage or cell nest, we may be free at least to accept the idea partially that the changes in the epithelium and the connective tissue both functional and anatomic can act as a basis for neoplastic changes. Thiem[17] reported 2 per cent of all carcinoma and 5 per cent of all sarcomas as the result of trauma. Loenhard[18] operated upon 500 malignant tumors— 477 carcinoma and twenty-three sarcoma—nine of the sarcomas and six carcinomas were due to trauma. Luckow,[19] in 1933, held that there was no dispute on the origin of a sarcoma from trauma. Campbell,[20] in 1941, reported two cases of carcinoma of the upper extremity due to trauma.

ESSENTIAL CRITERIA

Segond,[21] in 1907, Thiem, in 1909, and Lubarsch,[22] in 1912, established the following criteria: (1) The authenticity and adequacy of the trauma; (2) the previous integrity of the wounded part; (3) a reasonable time, relation from three weeks to three years or more in certain cases; (4) continuity of symptoms of the injury with those of the tumor; (5) microscopical or other proof of the existence and histological type of tumor, and (6) the location of the tumor at the point of injury.

Ewing[23] states that the important effects of trauma are: (1) Solution of continuity, minute and gross; (2) separation of cell groups and tissue masses, as of skin, gland and bone; (3) necrosis of tissue; (4) confined hemorrhage requiring absorption or encapsulation; (5) accelerated regenerative processes with hyperemia, and new growth of specific cells, blood vessels and supporting tissue and (6) cicatrization. Rixford[24] reported a case of cancer of the breast in a boy age seventeen, caused by fall on a tree stump striking his right breast. Mastectomy was performed five months later with death four years later from metastasis. When the records were submitted to Ewing he reported, "There is no escaping from the conclusion that in all probability, if not a certainty, the boy presented a genuine case of traumatic mammary cancer." Rienhoff[25] states, "cancer in regard to origin and nature is not an isolated inscrutable problem but one in harmony with microbic-pathological processes in general; it has a

concrete cause, is understandable from the viewpoint of natural science and is explainable in mechanical terms. It is an infectious growth, the same as sarcoma. The cancer cell is a dualistic organism in constitution and action, consisting in a living filtrable virus and a fixed living body cell. A minute investigation of injured single cells and their mode of healing in subcutaneous and superficial situations. Sarcoma is presumed to develop in a comparatively short time after a trauma directly in an injured but still living and recoverable cell to which an invading agent has to get by a circuitous route."

Crile[26] says that "cancer does not occur in normal uninjured cells but occurs after prolonged, repeated injury and repair due to mechanical, chemical, bacterial, electrical or radiation factors. Thus the occasional sarcoma of the thigh which follows a single crushing injury of all the soft parts down to the bone, leaving the skin intact, might be explained as due to autosynthesis. It is significant that this sarcoma sequence occurs only after a heavy blow or extensive crushing. This is comparable to the incidence of sarcoma in bone following a single blow, or in tissues which have not been lacerated and have incurred no infection, there has been no interference with the fullest development of bio-electric currents." Inclan[27] reported eight cases of giant cell tumors of the bone. Case 5 gave a history of a fall on her right knee, after which she complained of swelling, pain and limping. Eight months later the patient fell again on the same knee causing increased swelling and fever and functional disturbance. Three months later the tumor was excised.

Case 6, a white woman, age thirty-five, fell on her left knee, followed by pain, swelling and functional limitation. The pain continued and two years later resection of the tumor of patella was carried out. Gordon-Taylor,[28] in his excellent paper on sarcoma of the muscles and the connective tissue spaces of the limbs, reports twenty cases personally treated and refers to nine seen in consultation. In one case the patient had been struck by a cricket ball on the forearm—sarcoma of extensor muscles of forearm. He also reported one doubtful case of rhabdomyosarcoma of the lateral aspect of the left forearm due to a bite. Leighton and Schmidtke[29] presented before the Western Surgical Association, in 1938, seventy-nine case histories of superficial cancers initiated by a single trauma, which were not biased by any medico-legal phase, concluded that "While no one believes that a trauma in itself produces a cancer, it apparently does set off something, and I believe from our clinical experience that in certain cases, it is the inciting cause or instigation of the cancer."

H. H. Davis,[30] in his discussion concluded, "thus, it is seen that evidence can be given on both sides of the question at least, it can be stated that such an etiology is not common. Trauma reveals more

FIG. *1.* Case 1. Roentgenogram showing infiltrating tumor without invasion of bones.

malignant tumors than it causes." Davis, in his excellent paper before this Association, in 1940, is more sceptical, his conclusions in part are: (1) A single injury to previously healthy tissue has never been definitely proved to cause cancer; (2) nobody has succeeded in experimentally producing a series of malignant tumors by trauma; (3) trauma reveals many malignant tumors, whether it can actually cause a malignant tumor is an unsettled point. Metz, in discussing the paper of Davis' reported that in 228,000 accidents in twenty-five years they had seen no case of tumor due to trauma. "In the large volume of negative evidence that can be produced, it would seem conclusive that a single trauma could not produce malignancy, and that this Association could best devote its efforts to aid further in the research on malignancy and help in seeking true cause. Until the true cause is actually found and malignancy can be produced experimentally, we should not hesitate to admit that we do not know the

cause of cancer." In the same discussion, Nilsson reported four cases and Kreuscher two cases due to trauma.

CASE REPORTS

CASE I. Mrs. E. Z., age sixty-four, in October, 1938, severely traumatized her right hand and forearm in a washing machine wringer. As the
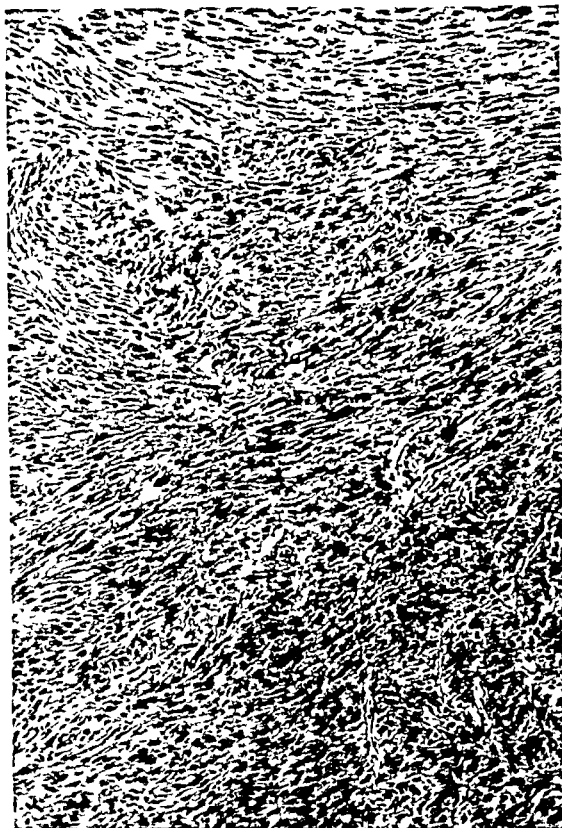
FIG. 2. Case I. Photomicrograph showing fibrosarcoma arising from tendon sheath.

wound did not heal in eight weeks a skin graft was done by Dr. Werra at the Waukesha Hospital. In January, 1939, she noticed a tumor on the back of her right wrist, which grew to the size of a hen's egg in one month. On February 10, 1939, the tumor was excised by Dr. M. J. Werra. Histological diagnosis by Dr. L. J. Van Hecke: fibrosarcoma. This was followed by six x-ray treatments but the tumor recurred in two months. On May 4, 1939, a radical excision was carried out followed by x-ray as she would not consent to amputation. A recurrence took place on September 1, 1939. When I first saw her September 7, 1939, there was a scarred area 8 by 10 cm. on the dorsum of the right forearm 3 cm. above the wrist. In this area were

four irregular rounded tumors 2 by 3 cm. in diameter and elevated about 1 cm. covered by atrophic skin adherent to the radius. The patient was admitted to Columbia Hospital September 8, 1939.

FIG. 3. Case II. Roentgenogram showing malignant tumor in distal end of right femur.

Radiographic diagnosis by Dr. S. A. Morton: Chest negative for metastasis, no evidence of bone disease, soft tissue tumor, right forearm on wrist. (Fig. 1.)

Operation on September 9, 1939, consisted of amputation of the right arm 3 cm. above the epicondylus lateralis humeri, which was followed by deep x-ray therapy. Histological diagnosis by Dr. G. H. Hansmann: Synovioma, right forearm (fibrosarcoma type). (Fig. 2.) The patient was discharged from the hospital September 27, 1939, in good condition.

On May 27, 1940, her condition was excellent with no evidence of recurrence. On June 10, 1940, metastatic sarcoma of the subclavicular lymph-nodes was found, and on August 13, hydrothorax. Paracentesis of the right chest was carried out four times from August 12 to September 7. The amounts of blood-tinged fluid obtained were 1,550 cc., 1,000 cc., 600 cc. and 1,000 cc., respectively. The patient expired on September 15, 1940. Anatomical diagnosis: Fibrosarcoma of right forearm with metastasis to right axilla, pleura- bilateral; hilar lymph-nodes.

CASE II. Mr. G. V., age sixty, three years ago when getting off a street car in the rain jumped over surface drainage and struck his right femur against a fire hydrant, which was followed by pain in the right knee for three weeks. Ten weeks ago pain recurred in the right knee from stopping sud-

denly while crossing the street; the pain radiated to the hip. He was treated by his family physician. His leg was placed in a plaster of Paris cast for five weeks without relief of pain. When first examined September 28, 1941,
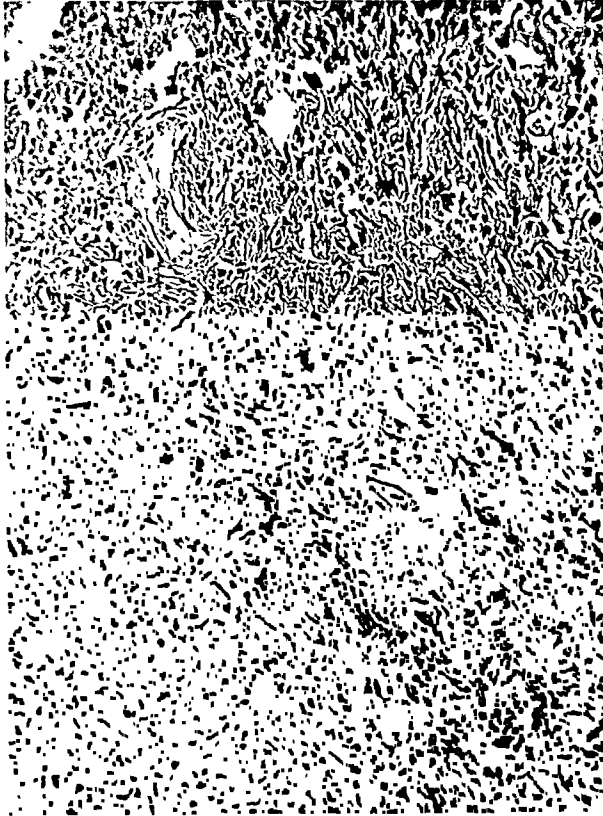
FIG. 4. Case II. Photomicrograph showing fibrosar-
coma of femur.

the skin of the right leg was atrophic, with capillaries dilated. There was pain on light pressure over semilunar cartilages which was increased on abducting the foot. Radiographic diagnosis by Dr. S. A. Morton: Malignant lesion lower end of right femur likely primary bone tumor. (Fig. 3.)

He was admitted to St. Luke's Hospital June 28, 1941. Radiographic examination of the skeleton showed no evidence of metastasis. On June 30, 1941, biopsy revealed fibrosarcoma, confirmed by Dr. Ed. Tharenger. (Fig. 4.) On July 9, 1941, a hip joint disarticulation was performed. He was discharged from the hospital July 26, 1941, in good condition. He refused to take deep x-ray therapy; at present there is no evidence of recurrence.

DISCUSSION

From a careful study of the literature it is seen that opinions are divided today as they were a century ago. With the enactment of compensation laws in the various States the medicolegal aspect of

malignancy has aroused a new interest in trauma. In recent years, success has followed efforts to produce tumors under strictly experimental conditions. Both carcinoma and sarcoma have been produced by x-ray in man and animals. Marie, Clunet and Baulot-LaPointe.[31] Fibiger[32] produced cancer with sudan III and Yamagiwa[34] and Ichikawa with coal tar. Baggs[35] produced mammary cancer in mice by stagnation of milk. Maud Slye[36] reported 14,000 autopsies performed on mice showing 1,301 indisputable neoplastic growths, of which fifty-one arose at the site of trauma. Pick[37] in studying the records of World War I found two cases of malignancy apparently due to trauma, the age group being an important factor. Behan[38] states, "Heredity, constitutional predisposition and other contributory causes are generally so associated in its origin that the possibility of a single indispensable agent acting as a sole exciting cause is universally doubted." Brosch[39] does not believe a single trauma can cause a tumor while McKendrick[40] states that there is no authentic case in the literature in which it can be proved beyond a doubt that fracture caused sarcoma. Since Schwann discovered the nucleus and nucleolus of the cell the cause and effect of injury to the cell has been studied by surgeons working in every branch of biology. Carrell[41] was the first to isolate in pure strains fibroblasts, osteoblasts, cartilage, epithelium, cancer cells, and leucocytes; and cone[42] cultivated *in vitro* peripheral nerves. Every one who has tried tissue culture *in vitro* appreciates the biological chemical and physical problems involved.

SUMMARY

1. During the past thirty years spent in the surgical clinic and pathology laboratory, two cases of sarcoma which fulfill the postulate of Segond as due to single trauma have been seen.

2. Until the cause of neoplastic diseases has been proved, I will conclude with the words of John Collins Warren,[43] "No young practitioner can be regarded as thoroughly equipped for surgical work who is not both a good Pathologist and an expert Bacteriologist. The confidence born of a knowledge of Pathology and Bacteriology enables him to assume grave responsibilities and to grapple successfully with the most complicated problems. It is from men thus equipped that we have a right to hope that the future masters of surgery are to be evoked."

REFERENCES

1. IMHOTEP. The Edwin Smith Surgical Papyrus. Translated by Breasted, J. H. Chicago, 1930. University of Chicago Press.

2. BICHAT. Anatomie Generale. 1801.

3. RASPAIL. *Bull. de Soc. Nat., Paris*, 19: 251, 1826.

4. SCHWANN. Frorieps Notizen, 1838.

5. MULLER, J. *U. d. fein. Bau. u.d. Formen d. Krankhaften Geschwulsten*, Berlin, 1838.

6. VIRCHOW, R. Die Krankhaften Geshwulsten. Berlin, 1863.

7. COLEY and HIGGINBOTHAM. *Ann. Surg.*, 98: 991, 1931.

8. RIBBERT, H. *Deutsche Ztschr. f. Chir.*, 47: 574, 1898.

9. VAN BERGMANN, E. *Med. Wchnschr.*, 1: 137–139, 1901.

10. ZEIGLER. General Pathology. Translated by Worthin. New York, 1908. Wm. Wood & Co.

11. LOWENSTEIN, B. G. *Bruns. Beitr. f. klin. Chir.*, 74: 715, 1911.

12. LEWY, R. *New York State Dept. of Labor. Med. Bull.*, No. 1, 1923.

13. MOCK, H. E. and ELLIS, J. D. *J. A. M. A.*, 86: 257–261, 1926.

14. VILLATA, G. *Policlinico (sez. chir.)*, 32: 434–439, 1928.

15. MASON, M. L. Carcinoma of the hand. *Arch. Surg.*, 18: 2107–2158, 1929.

16. DIETRICH, A. *Geschwukte als. Unfallfolge*, 3: 291–296, 1931.

17. THIEM, K. Handb. d. Infallerkrankungen. Ed. 2, p. 589. Stuttgart. 1909; *Monatschr. f. Unfallh.*, 19: 107–111, 1912.

18. LOENHARD, H. *Monatschr. f. Unfallh.*, 38: 45–450, 1931.

19. LUCKOW. *Ztschr. f. Arztl. Forth.*, 30: 617, 1933.

20. CAMPBELL, D. A. Carcinoma of the upper extremity. *Arch. Surg.*, 43: 803–810, 1941.

21. SEGOND, P. *Ass. Franc. d. Chir.*, 20: 745, 1907.

22. LUBARSCH. *Med. Klin.*, 8: 1651, 1912.

23. EWING, JAMES. Neoplastic Diseases. 4th ed. Philadelphia, 1940. W. B. Saunders Co.

24. RIXFORD, E. *Ann. Surg.*, 102: 814, 1935.

25. RIENHOFF, W. Principles and Foibles of Cancer Research. Baltimore, 1936. Waverly Press.

26. CRILE, G. W. Editorial. *Am. J. Surg.*, 55: 441–450, 1942.

27. INCLAN, ALBERTO. Surgical treatment of giant cell tumors of bone. *J. Internat. Coll. Surg.*, 3: 389–400, 1940.

28. GORDON-TAYLOR, GORDON. Sarcoma of muscles of limbs. *Brit. J. Surg.*, 28: 1–16, 1940.

29. LEIGHTON, W. E. and SCHMIDTKE, E. C. Single trauma as an etiological factor in carcinoma. *Tr. West. Surg. Ass.*, 48: 240–273, 1938.

30. DAVIS, H. H. Trauma and malignancy. *Am. J. Surg.*, 51: 641–652, 1941; *Tr. West. Surg. Ass.*, 48: 273–274, 1938.

31. MARIE, CLUNAT, BAULET-LAPOINTE. *Ass. franc. p. l'étude du cancer*, 239, 1912.

32. FIBIGER, Z. K. *Ass. franc. p. l'étude du cancer*, 13: 217, 1913.

33. FISCHER, B. *Deutsche path., Gesellsch.*, 10: 20, 1906.

34. YAMAGIWA and ICHIKAWA. *Japan path. ges., Tokio*, 1914; *Virchow Arch.*, 233–235, 1921.

35. BAGG. *J. Cancer Research* 9: 498, 1925.

36. SLYE, MAUD. *Ann. Surg.*, 93: 40–49, 1931.

37. PICK, L. *Med. Klin.*, 17: 416, 1921.

38. BEHN, R. J. Relation of Trauma to New Growths. Baltimore, 1939. Williams and Wilkins.

39. BROSCH, A. *Virchow's Arch. f. path. Anat.*, 162: 32, 1900.

40. MCKENDRICK. Quoted by Maxeiner. *Lancet*, 54: 757, 1934.

41. CARREL, A. Special Cytology. New York, 1928. P. B. Hoeber, Inc.

42. CONE, S. *Brit. J. Surg.*, 5: 524–558, 1918.

43. WARREN, J. C. Surgical Pathology and Therapeutics. Philadelphia, 1895. W. B. Saunders Co.

MASSIVE INTESTINAL RESECTION

E. P. COLEMAN, M.D. AND D. A. BENNETT, M.D.

CANTON, ILLINOIS

THE term, "massive intestinal resection," is generally accepted as referring to resections of the intestine measuring 200 cm. (6 feet 7 inches) or more. The mortality in such resection is exceedingly high and, therefore, the fatal cases are not recorded and the successful cases reported in the literature are comparatively few. Our interest in the subject is based upon experience in two cases, one of which was reported before the Western Surgical Association in 1940. There has been a sufficiently long follow-up period in both cases to warrant a statement at this time regarding the merits of the operation.

ANATOMY

Anatomists differ as to the length of the small intestine, Gray giving it as 20 feet 6 inches, Rost as 18 to 26 feet, with an average of 19 feet, and Treves an average of 22 feet 6 inches for the adult male and 23 feet 4 inches for the adult female. Beneke stated that for every 100 cm. (3 feet 3½ inches) of body length there were 387.5 cm. (12 feet 9 inches) of small intestine. The intestine is said to be longer in vegetarians than in meat eaters, as noted in the Russians and Italians who live on a bulky vegetable diet and in the Japanese whose diet consists mainly of rice.

PHYSIOLOGY OF DIGESTION

According to Rost, the small intestine secretes enterokinase which activates trypsin, erepsin which reduces albumoses and peptones to crystalline substances, lipase, nuclease, lactase and carbohydrate. Water is quickly absorbed in the upper part of the small intestine. Carbohydrates are absorbed as monosaccharides, and fats when converted into a soluble form as by the action of fat-splitting enzymes and bile. The absorption of food is practically completed in the small intestine. The jejunum absorbs more fluid and sugar, while the ileum absorbs more proteids and fats. A study of the medical literature indicates that a resection of the small intestine beyond 200 cm. (6 feet 7 inches) is quite likely to result in

serious metabolic disturbances, which disappeai, however, with the establishment of compensatory hypertrophy.

Experimental work on dogs has indicated that the rate of intesti-
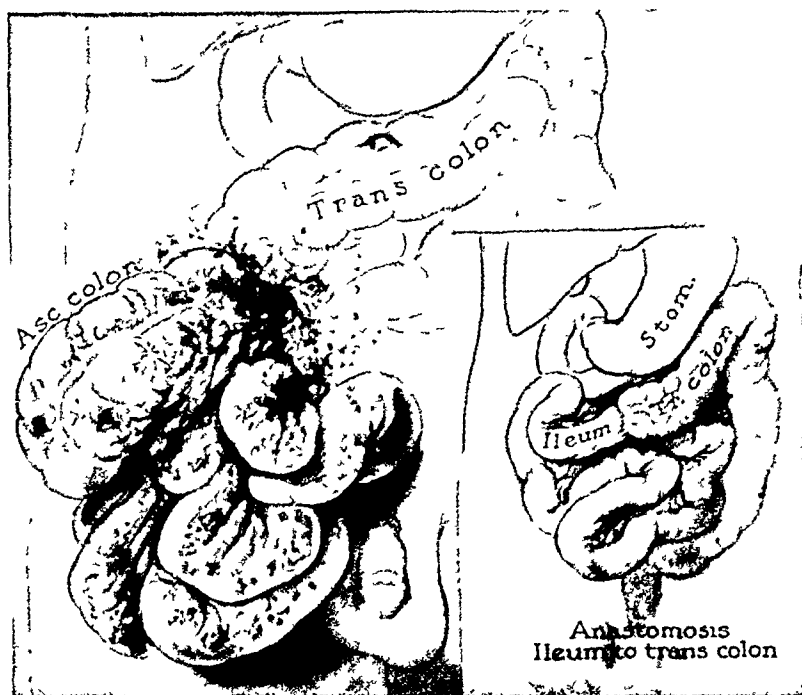
FIG. 1. Diagram of wound when patient was first seen. Insert: Postoperative appearance of anastomosis

nal absorption above the site of resection is twice that of normal. The compensatory reaction, therefore, occurs entirely above the resection and there is no evidence to indicate that the colon or intestine distal to the resection aids in this substitution. In resection of the jejunum there is an increase in pancreatic secretion, but in general after such resections the remaining intestine works more efficiently in the absorption of carbohydrates and does its poorest work in the absorption of fats. Consequently, any patient who has been subjected to a massive intestinal resection will do best on a high carbohydrate diet and with a limited intake of fats.

REVIEW OF THE LITERATURE

The earliest recorded case of resection of a large amount of intestine is that of Koeberlé (1881) in which 205 cm. (6 feet 9 inches) of small intestine were removed from a woman, aged twenty-two,

for multiple strictures. Recovery was followed by no digestive disturbances.

In 1896, Ruggi reported a resection of 330 cm. (10 feet 10 inches)

FIG. 2. Postoperative x-ray, showing short loop of intestine remaining, also several shot still present.

in a boy, aged eight, who made a good recovery. The same year Fantino recorded the resection of 310 cm. (10 feet 2 inches) of ileum from a man, aged sixty, who had a large inguinal hernia with volvulus of the intestine and gangrene. He made a good recovery and had no complaints when seen one year later.

In 1900, Hayes resected 255 cm. (8 feet 5 inches) of ileum from a boy, aged ten, who recovered, but suffered from occasional diarrhea and vomiting and developed chorea. In 1901, Lauwer reported the resection of 265 cm. (8 feet 9 inches) of small intestine from a woman, aged sixty-five, for ventral hernia with intestinal obstruction from adhesions. She recovered.

In 1907, Edward Staehlin recorded a resection of 10 feet 2 inches of small intestine, and Littlefield, in 1919, resected 13 feet of the ileum with a good result. In 1923, Pirie Watson reported a resection of 7½ feet of the ileum three years previously, with the patient not only in good health but free from nutritional disturbances. In the

same year Jacob Sarnoff, of Brooklyn, reported a case in which he not only resected 15 feet of small intestine but also did a hysterectomy. The postoperative course was complicated by a partial

FIG. 3. Stomach fifteen minutes after barium meal; transverse colon is beginning to fill.

intestinal obstruction and fecal fistula but the patient eventually made a good recovery.

The longest resection on record is in a case of Von Brenner, in which 18 feet of small intestine was removed. Four other cases averaging 400 to 540 cm. (13 to 18 feet) are those of Gheden (534 cm.), Nigrisili (520 cm.), Axhausen (475 cm.) and Pouchet (400 cm.). In each instance the patient recovered from the operation but died shortly thereafter.

CASE REPORTS

CASE 1.   This case was reported in detail by the authors before the Western Surgical Association in 1940.

Mrs. E. L., aged thirty-eight, para 3, was seen at the Graham Hospital, Canton, on the morning of July 6, 1939, with the complaint of severe abdominal pain. She had had many previous attacks of abdominal pain and

FIG. 4. One hour after barium meal. Colon is filled. Note exceedingly short length of jejunum between duodenum and site of anastomosis at transverse colon.

her appendix had been removed without relief of the symptoms. On examination there was a sausage-shaped mass, somewhat movable, extending from the right upper quadrant to the pelvis. A tentative diagnosis of paralytic ileus produced by a large retroperitoneal tumor was made. Because of an elevation of temperature the possibility of an infected right kidney was considered. The insertion of a Levine tube and Wangensteen suction gave no improvement, so operation was decided upon.

On opening the abdomen a large blue mass, too large to be delivered, appeared covered with peritoneum. The incision was enlarged, the mass delivered and found to be a thick, rubbery piece of intestine starting as an intussusception of the jejunum to 10 cm. (4 inches) from the ligament of

Treitz and involving a part of the ileum. The mass was about 15 cm. wide by 32.5 cm. long, and yet the mesentery was long enough, in spite of the edematous dense tissue, to permit the mass to be brought outside the
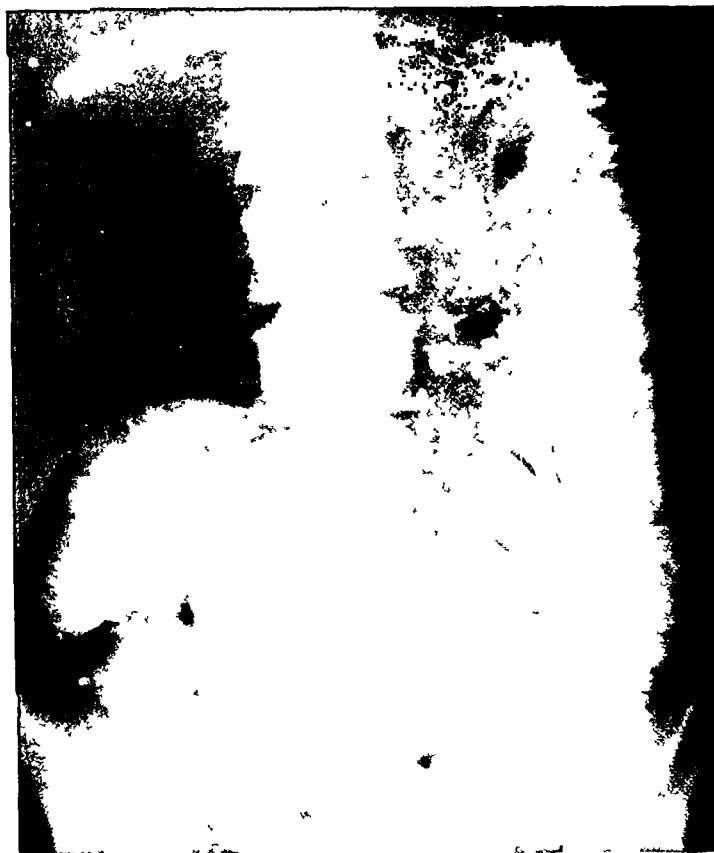
FIG. 5. Barium enema showing increased diameter of small intestine proximal to anastomosis.

abdominal cavity. Attempts to reduce the intussusception were futile, so the entire tumor was resected. Examination of the specimen showed that it consisted of nine layers of small intestine, that is, the intussusception was quadruple or "compounded" four times. It was 205 cm. (6 feet 10 inches) long. There were no adhesions present between the layers of the intussusception and no evidence of tumor, polyp or other cause could be found. There were large areas where the entire thickness of the wall was gangrenous, and the invaginated tissues were very edematous due to the long period of circulatory interference. The patient had a prolonged convalescence, leaving the hospital at the end of six weeks. She has been followed for three years. Roentgenogram of the stomach following barium meal revealed normal findings. She has no metabolic disturbance such as might be expected from the removal of so large a portion of the intestine.

CASE II. H. E., aged eighteen, entered the Graham Hospital on April 17, 1940, with a shotgun wound of the abdomen, accidentally inflicted while hunting. He was in severe shock and complained of abdominal
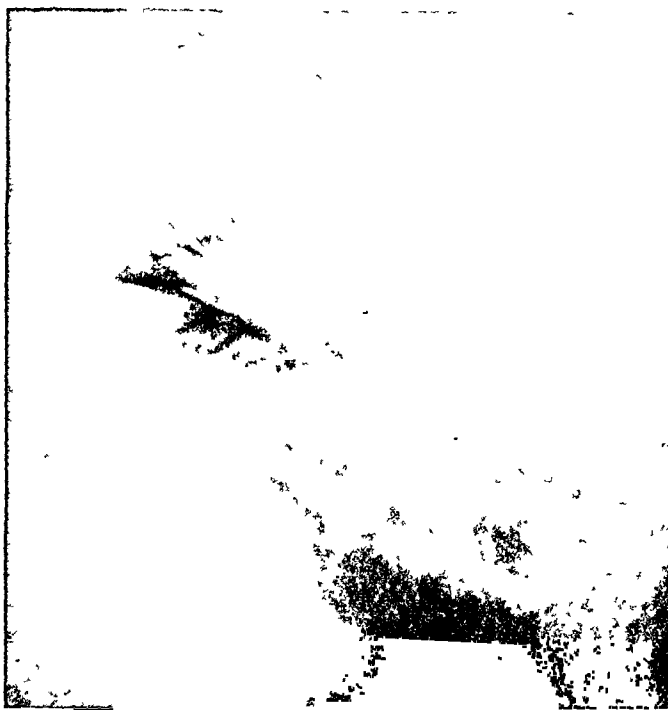
FIG. 6. Postoperative scar.

pain. In the right lower quadrant of the abdomen there was an opening 8 by 5 inches through which the intestine eviscerated. The eviscerated portion, which was bloody, dirty, perforated with shot and burned with powder, included the cecum, right half of the transverse colon, and many loops of ileum. A portion of the abdominal wall located over McBurney's point and above, measuring 6 by 4 inches, had been completely shot away.

The first impression was that the boy's condition was utterly hopeless. Nevertheless, sterile warm packs were placed on the eviscerated intestines, intravenous adrenal cortex was given, and intravenous glucose and saline started. He was typed for transfusion and found to be type 3. His father was type 4; however, 500 cc. of citrated blood was obtained from the father and added to the intravenous fluids. In about two and one-half hours he had recovered from the shock to such an extent that his temperature was 98°F., pulse 104, respirations 26, and blood pressure 95/65. His facial expression had changed from that of a moribund patient to one of mental alertness, and the transfusion had restored his color to some extent. It was thought that perhaps with a resection of a small amount of intestine the patient might have a chance for recovery.

Another transfusion of 500 cc. of citrated blood was started, and he was anesthetized with ethylene and ether. The gunshot wound was extended by an incision upward from the center of the wound, another one down along the right rectus muscle. Blood-stained fluid was present in the abdomen and considerable fecal matter had spread over the entire area. The damaged intestines were wrapped in sterile moist towels and the abdominal wall carefully cleansed with soap and water. No other skin preparation was carried out.

After redraping the wound, an examination was made to determine the extent of the intestinal damage. Since there were numerous shot lacerations of the bowel, both the intestine and its mesentery were damaged beyond any hope of repair, so the ileum was withdrawn from the abdominal cavity with the hope of finding an undamaged portion. He had been shot with a 12 gauge shotgun, the shell of which contained about 380 No. 6 shot, which will give some idea of the damage inflicted. Finally when the abdomen had been almost eviscerated, uninjured intestine was delivered which seemed to be the jejunum. Once more it appeared as though the condition must be hopeless, but as the patient's pulse was good and there was no place to stop, the small intestine was divided just proximal to the first shot perforation and all intestine distal to it was removed. Included in the resection were the remaining portion of the small intestine, the cecum which was badly lacerated, the ascending colon and hepatic flexure, which had only a few perforations, and the first half of the transverse colon, which had been practically torn in two. The end of the resection was just beyond the middle of the transverse colon. The mesenteric bleeding was controlled with hemostats and an end-to-end anastomosis was done between the small intestine and the transverse colon in two layers, the inner one of No. 00000 catgut and the outer layer of fine silk. The mesentery was sutured and the portion of the peritoneal cavity most extensively contaminated was irrigated with normal salt solution. The skin, fascia and muscle were carefully débrided until normal tissues were reached. The peritoneum could be closed satisfactorily, but the muscle and fascia had been so extensively removed that only the upper and lower angles could be sutured. An area of 5 by 4 inches of skin was left open and the defect filled in with a pack of sterile vaseline gauze, such as is used in the Orr treatment of osteomyelitis. The patient was returned to his room in surprisingly good condition, with a pulse of 126, respirations 24, and blood pressure 60/30.

The postoperative course was stormy and it is our belief that the patient was kept alive largely by transfusions twice daily. His highest temperature was 103.4°F. on the third postoperative day, and the highest pulse rate was 160 on the second day. The following morning he vomited, so a Levine tube was inserted and the Wangensteen suction apparatus applied. It was necessary to keep the tube in place for fourteen days, because each time it was removed the vomiting recurred. There was a considerable amount of serous discharge around the vaseline gauze pack; on the twelfth

day the pack was removed and replaced with a smaller one of the same type. He had no bowel movement until the third day when an enema was returned with a brown liquid and flatus. Following this, an enema every second day was returned with a brown liquid bowel movement. After the Wangensteen suction was removed, he was given a liquid diet of fruit juice and carbohydrates, and began to gain in weight and strength. As soon as his food intake was increased he developed a diarrhea which was checked by restricting the diet to fruit juice and carbohydrates. He was dismissed from the hospital the tenth week, weighing ninety-seven pounds, a loss of forty pounds from his normal weight. The wound was in good condition, with an area, 2½ by 3 inches, still uncovered.

The resected intestine measured 14 feet 8 inches with 22 inches of colon, a total of 440 cm.

Eight days after leaving the hospital he returned complaining of recurrent abdominal pain associated with diarrhea and a loss of five pounds in weight. On inquiry it was found that he had returned to his work as a fisherman and had partaken of fried fish on several occasions. Small amounts of tincture of belladonna were given and he was returned to a diet of fruit juice and carbohydrates, with no fats excepting small amounts of butter and cream with other food. Within two days the diarrhea was controlled; the stools became somewhat more solid but were still very largely liquid. He was then given six feedings a day of rather limited quantity and soon began to gain weight. At the end of one month he was discharged from the hospital, with the wound healed, and an average of two soft or semi-liquid bowel movements daily. He was much improved in strength and his weight was 103 pounds.

He returned for a check-up three months later, in November 1940, weighing 117 pounds. His diet was restricted largely to carbohydrates with a small amount of finely chopped meat and very limited intake of fats. Vitamins were furnished artificially and with the fruit juice. The bowels moved three times a day and the movement was usually liquid but occasionally semisolid. He was gaining in strength and considered himself in good health.

Nonprotein nitrogen was 32 mg. per 100 cc. of blood, and the fasting blood sugar was 104 Gm. per 100 cc. The blood count was within normal limits as was the urinalysis.

Roentgenographic and fluoroscopic examinations were made of the gastrointestinal tract. It was interesting to note that the barium meal upon leaving the stomach appeared to go through one short loop of intestine and enter immediately into the transverse colon. Not more than two or three feet of small intestine remained, but in spite of this loss his condition seemed remarkably good.

The patient was last seen on May 5, 1942. Roentgenographic examination showed the stomach quite enlarged and it emptied in three and one-half hours. The jejunum was also larger in diameter than is normal and

emptied directly into the transverse colon. The jejunum had enlarged and apparently had taken over the function of the ileum, and under the fluoroscope appeared as only a single loop of bowel. The barium meal on leaving the duodenum passed through this loop and into the colon, and the loop did not appear to be more than three feet long.

The abdominal wall was quite solid. The scar was thick and had a keloid appearance, such as is commonly seen after an extensive burn. He was in an excellent state of nutrition and was able to do the heavy work of a fisherman. He stated that abdominal cramps followed the eating of fried or greasy foods but no diarrhea occurred.

Blood chemistry examination showed blood sugar 105 mg. per 100 cc. of blood and nonprotein nitrogen 22 mg. per 100 cc. of blood. Urinalysis and blood count were within normal limits.

SUMMARY

Experience with these two cases brings out the following facts:

1. Massive intestinal resection can be performed with favorable results even under extraordinary conditions, such as were present in the second case.

2. The pre- and postoperative care played an important part in the final outcome of both cases.

3. The second case verified statements made in the literature that the diet after such resection should be high in carbohydrates and proteins, but low in fat and cellulose.

4. The author's experience with two cases of massive resection with recovery indicates that even in what appears to be a hopeless situation it is worthwhile to make every effort to save life.

REFERENCES

1. Ruggi, G. *Policlinico (sez. chir.)*, 3: 49, 1896.
2. Staehlin, Edward. *Ann. Surg.*, 45: 49, 1907.
3. Watson, Pirie. *Chir. Soc. Edinburgh*, 37: 164–183, 1922–1923.
4. Sarnoff, Jacob. *Ann. Surg.*, 78: 745, 1923.
5. Coleman, E. P., Maguire, R. H. and Bennett, D. A. *Am. J. Surg.*, 53: 340–344, 1941.

BRACHIAL PLEXUS BLOCK ANESTHESIA
OF THE UPPER EXTREMITIES*

R. A. GRISWOLD, M.D.†   AND   WILLIAM H. WOODSON, M.D.

Professor of Surgery, University   Resident, Louisville General
of Louisville School of   Hospital
Medicine

LOUISVILLE, KENTUCKY

THERE have been few recent reports in the American litera-
ture on the use of brachial plexus block anesthesia. The im-
pression obtained from conversations with many leading
surgeons is that the percentage of unsatisfactory anesthesia is so high
in most hands that this procedure is used only by a few who have
perfected the technic. The procedure most generally used has been
the supraclavicular route as described by Kulenkampff, quoted by
Labat.[1] Previous personal experience with this older technic was
unsatisfactory and the procedure was abandoned until Patrick[2]
described a modification which has been eminently successful in our
hands. The method of Kulenkampff and Labat depended upon the
disposition of the solution at exact anatomical points on the plexus.
These points were difficult for the novice or even the expert to reach
with the tip of the needle, particularly in obese patients. Patrick's
modification depends upon laying a wall of anesthetic solution from
the first rib out to the deep fascia at right angles to the path of the
plexus as it passes about the rib. Exact localization is unnecessary, as
the solution diffuses through the fascial compartment occupied by
the plexus. This technic has in our hands reduced primarily unsuc-
cessful anesthesia to 3 per cent.

Premedication is of primary importance not only to allay the
patient's apprehension but to protect against procaine toxicity. A
barbiturate, usually sodium phenobarbital (.130 Gm. for 150 pounds
of body weight) is given hypodermically thirty minutes before the
block is started, or if time is pressing, one-half of the above dose is
given intravenously. The patient is placed on the table, supine, with
a thin pillow beneath the upper dorsal spine, the shoulder on the
involved side is depressed, the arms placed at the side and the head
turned toward the opposite side.

The surgeon determines by palpation, the midpoint of the clavicle, between the sternoclavicular and the acromioclavicular joints. An intradermal wheal of novocaine is raised ½ cm. superior to the midpoint of the clavicle. A No. 22 gauge needle three inches in length and not attached to the syringe is then inserted, inward, downward and backward, and roughly toward the second or third dorsal spine, until the first rib is encountered well posterior. If blood is encountered at any time, the needle is withdrawn and reinserted at a slightly different angle. The needle after encountering the first rib is withdrawn 2 to 3 mm. and with a glass syringe 10 cc. of 2 per cent novocaine is injected as the needle is withdrawn to the level of the deep fascia. It is then reinserted at a slightly different angle with the needle point more lateral and anterior but still contacting the first rib, and 10 cc. of solution is injected as the needle is withdrawn. The object in withdrawing the needle point is to enable permeation of the nerve trunks of the plexus. It must be recalled that the brachial plexus and the subclavian artery lie like meat in a sandwich between two fascial planes: in front, a layer of fascia extending across from

TABLE 1

			Simple	Compound
1. Reduction of Fractures:				
Colles's			2	0
Ulna and radius	U/3		4	0
	M/3		11	2
	L/3		4	1
Radius	Head		1	0
	U/3		1	0
	L/3		5	0
	Resection of head		3	0
Humerus	U/3		0	1
	M/3		1	1
	L/3		1	3
Metacarpals			1	2
Phalanges (multiple)			1	1
Navicular			1	0
Traffic elbow			0	1
Open reduction	Olecranon		4	0
	Metacarpophalangeal, joint		1	0
			41	12
11. Primary Tenorrhaphy:				
Biceps L/3				1
Triceps L/3				2
Forearm and wrist				7
Fingers				8
Hand				4
				22

Table i—(Continued)

	Compound
iii. Débridement, Irrigation and Suture of Lacerated Wounds.	
Elbow..	4
Forearm.	5
Hand ...	1
	—
	10
iv. Débridement, Irrigation and Suture of Gunshot Wounds:	
Forearm	2
Hand.	1
Humerus M/3	2
Fingers	1
	—
	6
v. Amputations:	
Arm M/3	1
Forearm M/3	1
	—
	2
vi. Miscellaneous:	
Ganglionectomy of wrist	1
Incision and drainage of abscess of web space (severe diabetic)	1
Excision palmar fascia (Dupuytren's)	1
Hemisection of hand	1
Removal of screws from graft on radius	1
Débridement human bite of hand	1
Excision tuberculoma of elbow	1
	—
	7

the scalenus anticus, and behind by a layer over the scalenus medius. By withdrawing the needle during the injection this compartment is traversed and a triangular wall of solution is deposited, based on the first rib with the apex of the triangle at the wheal.

The needle is reinserted some four to six times, always through the initial intradermal wheal advancing the needle point laterally and finally medially along the first rib from back to front. No attempt is made to encounter or provoke paresthesias. However, the patient is told to expect pain running down the arm especially to the little finger and to tell the surgeon. If such a pain is encountered, a single injection is made without moving the needle, directly into the plexus. There are anatomically no dangers laterally, but as the medial and anterior position is approached the subclavian artery must be missed by the needle point. The lower trunk of the plexus lies deep to the artery and must be injected to assure a perfect block. When the needle is inserted at its most sternal portion, it is seen to pulsate with transmitted beats from the subclavian artery. It is here that the final injection is made. Often the presence of the artery is

first made evident by blood exuding from the needle. If this happens, remove the needle and reinsert at a slightly different angle. At no time despite several entrances into the vessel has there been any hemorrhage from the vessel or any other untoward complication. The amount of 2 per cent novocaine was arbitrarily set at 60 cc., as the maximum dose for a 150 pound man without chancing immediate or delayed novocaine reactions.

The block is given with the surgeon wearing sterile gloves. Fifteen minutes should elapse before starting the operation after completing the injection. This period is used for the surgeon to scrub, don gown and gloves and prepare the field of operation. By this time anesthesia usually is complete.

The blocks reported in this series are 100 consecutive cases done in the Louisville City Hospital (University of Louisville School of Medicine) by the surgical resident staff from July, 1940, to October, 1941. A total of eighteen house officers contributed their cases. Several of them performed only one case and the largest number of cases by one man (W. H. W.) was twenty-nine. It is interesting that eighteen cases in this series represent the surgeon's initial case.

The cases selected for brachial blocks were those of trauma to the upper extremity. There were fifty-three reductions of fractures of which forty-one were simple and twelve compound. Of the simple fractures, eight had open reduction. The fractures ranged from multiple phalangeal fractures to a compound fracture of the upper third of the humerus. Twenty-two primary tenorrhaphies were done in the lower third of the arm, forearm, wrist, hand and fingers. Ten lacerated wounds of the elbow, forearm and hand were débrided, irrigated and sutured. There were six gunshot wounds of the arm, forearm, hand and fingers that were débrided, irrigated and sutured. Two amputations, one through the middle third of the arm and the other, the middle third of the forearm, were done. A miscellaneous group of seven cases consisted of: (1) ganglionectomy at the wrist; (2) incision and drainage of an abscess of the web space of the hand in a severe diabetic; (3) excision of palmar fascia for Dupuytren's contracture; (4) hemisection of hand; (5) removal of screws from graft on radius; (6) débridement of human bite of hand; (7) excision of a tuberculoma of elbow.

No attempt was made to measure the length of time that the anesthesia lasted, but the average length of 100 operations done under brachial block alone was sixty-seven minutes. The shortest operation required three minutes. This was an incision and drainage

of a web space abscess of the hand in a severe diabetic. The longest case required 195 minutes and was a tenorrhaphy on the finger flexors. As would be expected in a traumatic series such as this the males predominated the females 77 to 23. There were fifty-nine white cases as compared to forty-one negroes. The average age of the patients was thirty-two years. The youngest patient was an eight-year old white girl who had a reduction and cast for a fracture of the middle third of the ulna and radius, the procedure lasting sixty minutes. A total of five nine-year olds had forearm reductions or tenorrhaphies. The upper age limit was represented by two men aged sixty-seven, one of whom had a compound traffic elbow and the other a severe laceration of the forearm and cubital fossa. The former operation lasted 140 minutes and the latter 150 minutes.

Of the 100 cases three were initial failures. It is interesting to note that one of these patients had an excellent motor paralysis but had a poor sensory loss. One of these represented the first block attempted by a member of the house staff. The third failure also developed a small pneumothorax on the same side due to poor technic. A second case developed a 25 per cent pneumothorax with excellent anesthesia of the upper extremity. Neither of these complications were of any significance. Two patients had mild procaine reactions that were easily controlled with intravenous sodium phenobarbital. One of these had received no preoperative barbiturate. In one case cyclo-propane was used after fifteen minutes of successful block because of the unco-operativeness of the patient due to alcoholism. Only eight cases required some type of supplementary anesthesia because of the wearing off of the block anesthesia before completion of the operation. The shortest time a block lasted was sixteen minutes and the longest was eighty minutes before supplementary anesthesia was required. All cases have been followed in postoperative and fracture clinics and there has been no evidence of any delayed complications.

SUMMARY

1. A technic of supraclavicular branchial plexus block anesthesia is described and a consecutive series of 100 cases reported with three failures.

2. The operations performed under this type of anesthesia are presented.

REFERENCES

1. LABAT. Regional Anesthesia. Philadelphia, 1930. W. B. Saunders Co.
2. PATRICK, J. Brit. J. Surg., 27: 734, 1940.

THE TRANSVERSE PALMAR INCISION

LIEUT. COL. L. H. McKIM, R.C.A.M.C.

Officer in Charge Surgical Division, Debert Military Hospital, Canadian Army

DEBERT, NOVA SCOTIA

THE transverse palmar incision is not new. It has been in use in the Montreal General Hospital for at least thirty years. Its originator is not known. It was described by the author in 1926[1] and was published by him in 1928.[2]

The many advantages of this incision, and its practically total lack of disadvantages, are, I believe, sufficient excuse for bringing it to the attention of the profession.

Some of its more important advantages are: (1) Complete and adequate exposure in infections of the palm of the hand ("collar button," web or lumbrical space infections and midpalmar space abscess); (2) ease of combining with the lateral finger incision for suppurative tenosynovitis; (3) ease of enlargement to secure complete relief of tension, if necessary across the whole width of the palm; (4) complete freedom from danger of postoperative, or post-infective finger flexion deformity, similar to the Dupuytren's type; and (5) an almost invisible and totally painless scar.

Like all others in the treatment of hand infections, this incision should be made in a "bloodless field." This implies the use of general anesthesia. Insistence on these two elemental points should be unnecessary.

TECHNIC

The incision should not be made too far proximally. About one-quarter of an inch from the proximal transverse crease at the base of the finger is usually the correct site. The two fingers near whose bases the maximum lesion is present are slightly separated and a small incision is deepened until pus is encountered. A director is then passed transversely until the limits of the cavity are reached. This will be found to be superficial to all important structures such as tendons, blood vessels and nerves. The cavity may then be explored with the tip of the finger and all necrotic material wiped out with gauze. (Fig. 1.)

The frequent occurrence of palmar infections in conjunction with, or as a complication of, suppurative tenosynovitis of the flexor

sheaths of the fingers, makes necessary the description of the lateral finger incision. The ease of combining the two incisions has already been referred to.

FIG. 1.
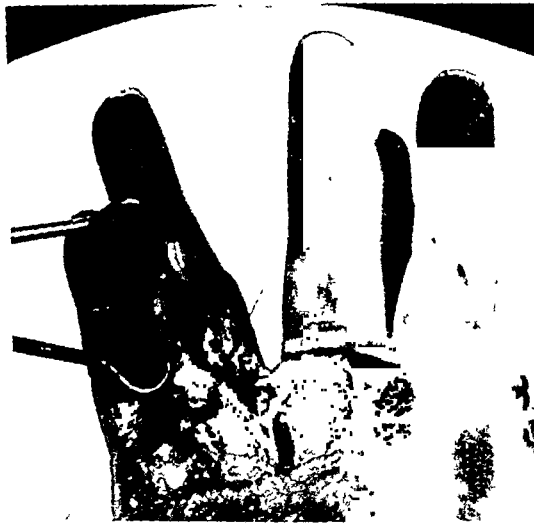
FIG. 2.

The principles on which the *lateral* finger incision is based are, curiously enough, almost identical with those in favor of the *transverse* palmar incision. The main one is the avoidance of placing a scar at right angles to a flexion crease.

The finger is flexed and the incision is made at a point just dorsal to the termination of the interphalangeal flexion crease. It should be begun on the lateral aspect of the proximal phalanx and continued distally and proximally as is necessary. Care should be taken not to damage the capsule of the interphalangeal joints. The incision should be carried out with the utmost care in the proximal direction and should not be allowed to approach any nearer to the palmar surface of the finger, but should be carried along the same plane to the web. When this point is reached, it is connected with the palmar incision by cutting through the web at a point exactly midway between the fingers and at right angles to the palmar incision. Failure to observe this latter point is about the only way damage can be done to the digital vessels and nerves. This is well shown in Figure 2 in which the combined incisions are seen with the vessels and nerves safely entering the anterior finger flap. The possibility of their damage by a careless "corner cutting" procedure should be quite evident.

The author will, he trusts, be pardoned for a word in defense of the much condemned "fish-mouth" incision which happens to be shown in the illustration. Practically all the unfavorable results ascribed to its use are due to its being made too far anteriorly. If it is made quite close to the nail, the question of a scar on a pressure-bearing surface will not arise.

Use of the B. I. P. P. and liquid paraffin and curtain drainage method as described in the original article[2] and in later publications[3,4] has, over a period of years, served to convince the Author of its superiority to all others, in the treatment of hand infections, and most particularly in connection with the incision just described.

REFERENCES

1. McKim, L. H. Infections of the hand. Read at Annual Meeting of American College of Surgeons, Montreal, October, 1926.
2. McKim, L. H. Infections of the hand. Canad. M. A. J., 18: 17–22, 1928.
3. Gurd, Fraser B. and McKim, L. H. The use of B.I.P.P. and liquid paraffin in the treatment of wounds. Am. J. Surg., 51: 584, 1941.
4. Gurd, Fraser B. and McKim, L. H. B.I.P.P. and liquid paraffin treatment of war and civilian wounds. Ann. Surg., 113: 987, 1941.

IMPACTION OF FRACTURES BY LARGE PRESSURE TONGS

ARTHUR R. METZ, M.D.,
RAYMOND HOUSEHOLDER, M.D. AND JAMES F. DEPREE, M.D.
Attending Staff, Wesley Memorial Hospital
CHICAGO, ILLINOIS

THERE are some types of fractures that can best be treated by forcing and molding the fragments back into position. The most common are the fractures of the os calcis and the proxi-
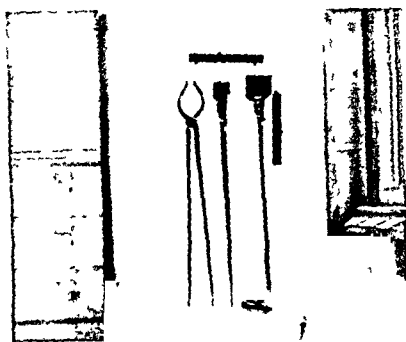
FIG. 1. Large pressure tongs three feet long; hinge seven inches from blade end; blades padded with felt.

mal end of the tibia. Other fractures may be treated in this manner but they are not common.

The os calcis is usually fractured by a fall from a height which produces a displacement upward with a broadening of the body of the bone so that it impinges on one or both malleoli. If this broadening is not corrected, there is a persistent deformity which causes pain on walking. By reversing the mechanism that produced the fracture the fragments can often be molded and forced into their normal position and will remain so if the foot is placed in a cast.

A quick and easy way to accomplish this maneuver is by the use of large pressure tongs with padded blades as shown in the illustration. A firm squeeze on the os calcis just below the malleoli combined with traction will often give a very satisfactory reduction. The tongs are made large with long handles and padded blades so that considerable quick pressure can be used without much effort, and no
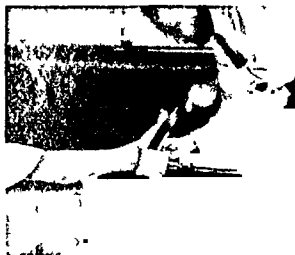
FIG. 2.   FIG. 3.   FIG. 4.

FIG. 2. Apply firm pressure below malleoli to squeeze bone into position using counter-traction behind heel and against sole.

FIG. 3. Tongs can be applied to outside of cast to make gentle pressure while cast is hardening.

FIG. 4. Tongs are used over the head of the tibia to push the fragments together.

FIG. 5. Fracture of os calcis; before and after use of pressure tongs.
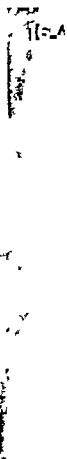
FIG. 6. Fracture of tibia; before and after use of pressure tongs.

damage is done to the soft parts. A small pad of felt may be placed just below the malleoli after reduction and incorporated under the cast. Slight pressure may be applied to the cast over the felt pads with the tongs while the plaster sets so as to insure a snug fit over the sides of the os calcis below the malleoli.

The manipulation is best carried out under an anesthesia, such as morphine-scopolamine, as soon as possible after the injury and before much swelling has occurred.

The cast should extend from the toes to below the knee. In more extensive fractures it may be necessary to extend the cast onto the thigh with the knee flexed to a right angle so as to keep the tendon Achilles relaxed.

Some fractures of the proximal end of the tibia and other bones are of such a type that a firm quick squeeze will force the fragments into their normal position and they will remain so without any other treatment than fixation.

The first large pressure tongs we used were hammered out by a blacksmith in 1928 and have been used frequently. We present the idea now as another method that may be used to advantage in the treatment of a selected group of fractures.

A PLEA FOR CAREFUL EXAMINATION AND EARLY EVALUATION OF TRAUMATIC CASES

E. PAYNE PALMER, M.D.

PHOENIX, ARIZONA

THE traumatic surgeon should have an abundance of time to give to each injured patient. Every traumatic case should be carefully examined under proper surroundings as soon as the patient's condition will permit. Even the slightest trauma should receive careful consideration, since minor injuries may not only produce disability and deformity but also end far too often in death. An injured person is entitled to a most careful and complete examination with all pertinent findings being recorded. The best obtainable consultants should be brought into the case early, when group consultation is desirable. This will be fairly frequent, as many traumatic patients are too seriously injured for a lone surgeon to contend with. A traumatic patient carefully examined with the findings recorded is more certain of proper treatment, and consequently has better chances for a satisfactory recovery.

In no other field in medicine does so much depend upon repeated x-ray examination for diagnosis and correct evaluation. Skilled and discriminating x-ray interpretation, based upon faultless technic, can do much for a better solution of the many problems involved in the evaluation of disability. Roentgen examination should follow the clinical investigation, unless bone injury can be absolutely excluded by the report of the accident and the clinical findings; otherwise, regardless of the extent and nature of soft tissue damage, x-rays should be taken early soon after the accident. As fractures frequently are unrecognized, an early evaluation may be incorrect. No guessing is to be tolerated in any case.

The difficulty in evaluating lower back disability, that greatest bugbear of industrial practice, arises out of one, or it may be all, of four failures on the part of the practitioner: failure to know minutely the anatomical structure of the lower back and the anomalous variations so frequently found; failure to obtain a detailed and correct history of the mechanism of the injury; failure to have an adequate

x-ray examination; failure to guide carefully and gradually the rehabilitation and re-education after a period of immobilization.

All of these four points are essential. In particular the consulting surgeon on traumatic cases is often astounded at the rashness with which both positive and negative conclusions are drawn from inadequate x-ray examinations. The majority of cases have only anteroposterior and lateral views, which are not sufficient; many instances are seen in which only anteroposterior flat films are made and conclusions are drawn from these alone. Not only are oblique views necessary, in addition to anteroposterior and lateral views, but many injuries to the lumbosacral area should have films taken in upright as well as supine posture. In addition the lumbosacral area requires films taken at a different angle from the remainder of the lumbar spine; consequently, the usual films of the lumbar spine as a whole are not adequate for the lumbosacral region. Stereoroentgenograms are frequently indicated. Finally, study and interpretation by a trained and qualified roentgenologist is most desirable, unless the industrial surgeon is so qualified.

In every disabled traumatic case there are factors which are immeasurable, imponderable and intangible. The traumatic surgeon owes it to his patient to see that an accurate estimate of his disability is made early. Not only has the traumatic surgeon a serious duty in estimating the disability sustained by a patient, but also a definite responsibility toward the insurance carrier; therefore, he must be strictly impartial. The end results deserve first consideration. A diagnostic or therapeutic error may be responsible for prolonged treatment and for a high degree of permanent disability with all its implications for the injured person as well as for the insurance carriers.

The traumatic case is not a machine to be repaired but a human being to be helped. No two persons are alike in thought or action, in temperament, personality or emotional reactions. Therefore, the examiners must listen to the patient attentively, observing him carefully to obtain a correct estimate of his attitude toward recovery and his desire or willingness to return to work.

Though several prominent traumatic surgeons—Keisler, McBride, and others—present very practical methods for evaluation of traumatic cases, there is no substitute for training, experience and sound judgment.

Whatever method may be used to evaluate a disability, it is usually found that the simpler the method, the better the results.

The necessity for some guide, however, in determining the average period of incapacity for given clinical conditions is apparent. Studies of nonlitigation cases furnish a useful basis for establishing average periods of incapacity, but these averages must be considered as the irreducible minimum.

The attitude of the physician treating the traumatic patient will materially influence the mental attitude of the patient. Patients told by their attending surgeon that their injuries are unusually serious will have an unfavorable outlook toward recovery, as compared with the patient who has been encouraged and told that he will make the best recovery possible. Recovery may be retarded when the attending surgeon overemphasizes caution. Patients in a ward with dissatisfied traumatic patients should be moved to new quarters whenever this condition is discovered. Again, patients in contact with dissatisfied friends and relatives will be influenced unfavorably toward recovery. Everyone about the patient should be asked to help build up a healthy attitude toward recovery in order that an early evaluation can be made. Also forewarn against discouragement upon resumption of work and encourage perseverance. The compensation patient, paid for his disability, is likely to prolong his complaint of disabling symptoms, whereas the noncompensation patient will make an effort to return to work as soon as possible. He will often put up with the inconveniences occasioned by an injury, whereas the compensation patient will expect to be rid entirely of all inconvenience before he returns to work.

An accurate and detailed history is needed not only of the accident in question but of any diseases or former accidents; only in this way will a definite visual reproduction of what actually took place be reproduced in the mind of anyone hearing or reading the description.

Regardless of whether the case is compensable, the traumatic surgeon and his consultants should evaluate the injuries and their results as early as possible. They should determine the direct and proximate result of the injury, and the length of time necessary to relieve the condition. They should estimate the probable outcome; how long it will take the injury to reach its final stage of healing; whether the disability will be temporary, with complete restoration of function; or permanent; and finally, what per cent of the permanent loss of function has resulted from the injury. Experience proves that many traumatic cases have definite disability not related to the injury; nevertheless, symptoms resulting from such disability often are attributed to the accident by the patient. Frequently, he may

believe such is really true; therefore, the physician must determine how much of the disability developing subsequent to an accident is actually due to that accident. This determination can be made only when competent medical observation has decided how much of the disability is the natural result of the disease and how much added disability has been produced by the accident. The rational approach would seem to be to attempt to decide what the natural progress of the disease would have been without the accident and then to add the effect of the accident to this. A systematic, careful, detailed and intelligent study of each individual case is the only procedure which will bring satisfactory results. Each case is a problem unto itself and should be studied from every aspect, historical, anatomical, physiological, pathological and psychological, utilizing every resource of clinical and laboratory analysis.

The traumatic surgeon and consultants, therefore, when evaluating traumatic cases, should obtain a history of the accident, the progress of the case up to the time of evaluation, and the symptoms complained of at the time of the examination; all of this should be recorded. Then they should make a detailed and painstaking physical examination, carefully recording the complete findings. Finally, they should examine such laboratory records, including roentgenograms, as are at hand and order such other examinations as are deemed advisable to secure the necessary data.

Many problems must be considered in the evaluation of traumatic cases. Unfortunately, the period of temporary disability as well as the percentage of partial permanent is influenced by the factors of compensation and noncompensation. The mental attitude of the injured person toward his disability and his inclination to return to work are strong factors in each individual case. The traumatic patient's concept of himself and the outer world frequently undergoes a drastic change unless an effort is made to bolster his morale. The traumatic surgeon will find that the proportion of patients presenting mental symptoms of etiological significance during their illness is much larger than is generally believed. Since, as is well known, purely emotional causes, if severe and prolonged, may produce structural changes, the physician should guard against a traumatic neurosis in his patients, using every means to prevent it. The onset of symptoms indicating this condition means there is reason for haste in evaluating the disability. The treatment of traumatic neurosis, so-called, in the acute stage is prophylactic.

Therefore, the earlier the evaluation, the quicker and more certain will rapid recovery occur.

One must remember that anatomic repair does not mean functional restoration is fully established. Sufficient time must elapse to allow the ordinary consequences of trauma to disappear by natural body means. The injured person need not wait, however, for the maximum adaptation, but through re-education to altered conditions, return to work and endeavor to obtain maximum efficiency. One with a physical handicap must, therefore, develop new habits of use so that improvement through adaptation is not final until the injured part can be used without conscious effort. The attending surgeon should foresee such conditions and explain to the patient what is to be expected, thereby preventing discouragement and dissatisfaction with the evaluation of the disability.

CONCLUSIONS

1. After taking part in more than 1,200 industrial consultations, it is my opinion that traumatic patients require a very careful examination for a correct diagnosis, to get the best treatment possible, to reduce both temporary and permanent disability and to obtain an early evaluation.

2. A consultant or group consultation is advisable for correct diagnosis and exact evaluation. Such service should be assigned to the most thoroughly trained and experienced traumatic surgeon and specialist in other fields of medicine having a knowledge of traumatic cases.

3. Properly executed roentgen examination should follow the clinical investigation. Skilled and discriminating x-ray interpretation by a thoroughly trained and experienced roentgenologist is most desirable as an aid to diagnosis and correct evaluation.

4. The mental attitude of the traumatic patient toward his disability and inclination to return to work is materially influenced by a number of factors, especially by the attitude of the attending surgeon. Giving expectation of improvement will result in a more rapid recovery and an earlier evaluation.

5. An early determination of the degree and type of the trauma is a prerequisite to the early and correct evaluation of disability.

6. Early evaluation insures the elimination of uncertainty on the part of the patient, a more speedy recovery, and an earlier return to work.

ADJUSTABLE TRACTION SPLINT FOR UPPER OR LOWER EXTREMITY

CHARLES S. VENABLE, M.D.

SAN ANTONIO, TEXAS

Diplomate of the American Board of Surgery; Chief of Staff, Nix Hospital; Attending
Surgeon, Santa Rosa Hospital

DUE to limitations and priorities on metal necessarily used in making Thomas or Murray-Jones splints, etc., I have devised this adjustable wooden traction splint applicable for splinting
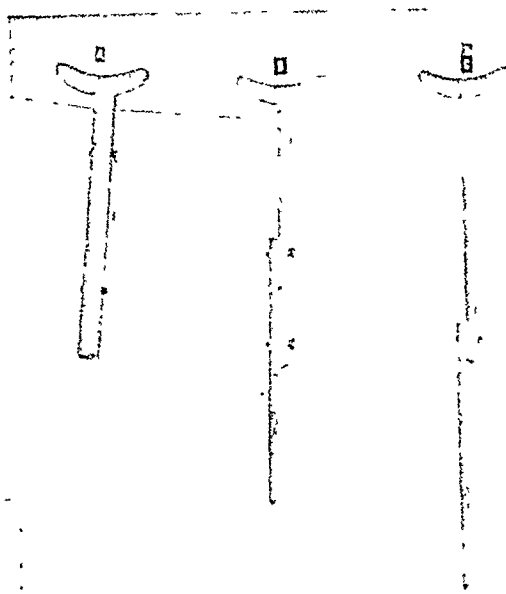
FIG. 1. A, splint closed; B, splint extended for traction, upper extremity; C, splint extended for traction, lower extremity. Because of tongue-groove principle, splint needs only one bolt.

either extremity in traction. The one piece fixed to the "crutch" is grooved while the movable piece is tongued so that it may slide (tongue and groove principle), from an over all of 25 inches to be locked by one or two bolts at any desired length up to 45 inches, when it is perfectly rigid against rotation as well as bending.

In applying traction to a fracture in the upper extremity the "crutch" is placed at the junction of the pectoral muscle and chest wall with a hitch about the base of the hand which is fixed over the
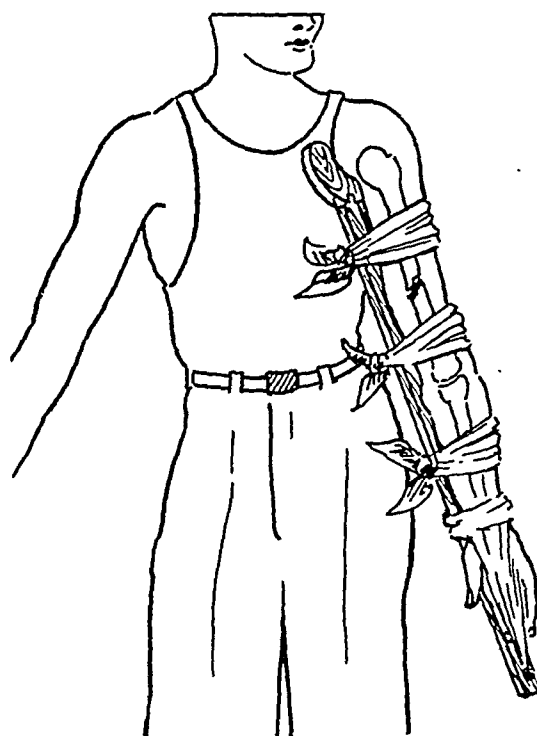
FIG. 2. Splint applied with traction and fixation in fracture of humerus. Note slight abduction with pressure of crutch against pectoral muscle. The tighter the traction, the greater the abduction.
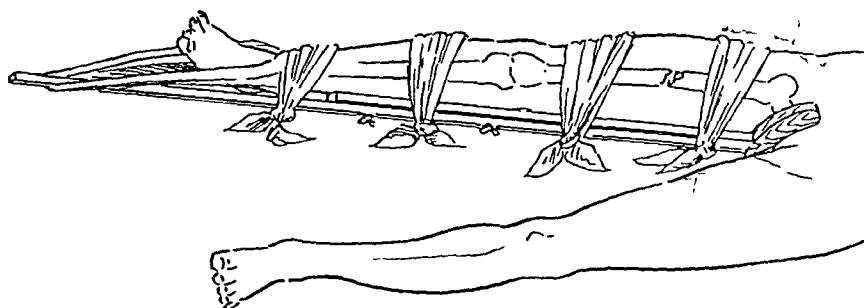
FIG. 3. Traction and fixation in fracture of lower extremity. Fragments are well mobilized for transportation.

distal end, which is notched, so that the greater the traction the more the abduction of the extremity. This position prevents "crutch" paralysis. A bandage, if available, or one or two handkerchiefs, etc., will stabilize the arm to the splint.

In fractures of the lower extremity the "crutch" is placed in the crotch, when with a hitch about the ankle sufficient traction is easily had. The limb is then stabilized to the splint with two or three ties of some sort.

When not in use it occupies some 26 inches of space and weighs 12 ounces. The object is to have a sufficient number of these splints available everywhere in order to carry out the slogan, "Splint 'em where they lie" in order to minimize trauma and stabilize fractures during transportation.

The American Journ of Surgery

EDITED BY THURSTON SCOTT WELTON, M.D.

January 1943

Gynecology . Obstetrics

Orthopedics . Neurosurgery

Gastrointestinal Surgery . Proctology

ARMY-NAVY PRODUCTION AWARD

A token of the unity of our work with that of many others striving towards the victory we all hope soon to achieve

Every member of the Davis & Geck organization regards the Army-Navy "E" pennant, now flying with the Stars and Stripes over our building, as a signal honor which we must and shall continue to merit

D & G SUTURES . . . THIS ONE THING WE DO

D&G Sutures with
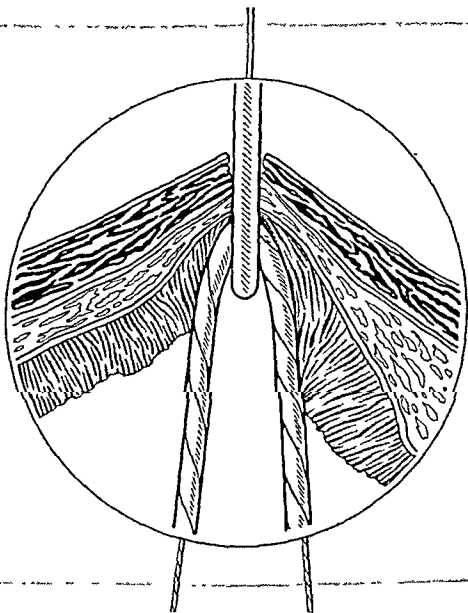
To MEET varied situations where minimized suture trauma is desirable, over one hundred D&G Atraumatic suture-and-needle combinations are now available. These range from delicate nerve sutures of size 8-0 silk to sturdy obstetrical sutures of size three catgut and include extremely fine strands armed with needles at both ends for convenience in certain procedures.

The practicability and convenience of these products has been demonstrated by a constant increase in usage. Davis & Geck, therefore, has extended the Atraumatic principle to a wide variety of products for use in general surgery as well as in the specialties.

The needles are of high quality steel, tempered against brittleness and resultant "snapping". Patented construction provides a sleeve of exceptional strength which will not bend or break; the method of affixing insures positive anchorage to the suture material—it cannot pull out. All curved needles have a flattened
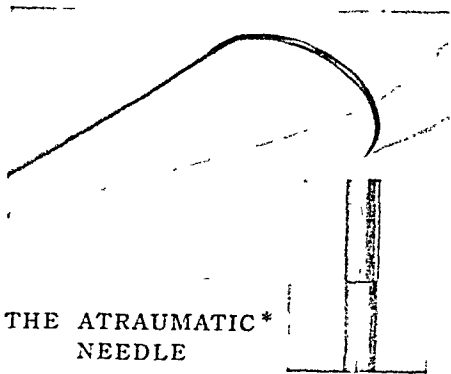
THE ATRAUMATIC* NEEDLE

Atraumatic Needles
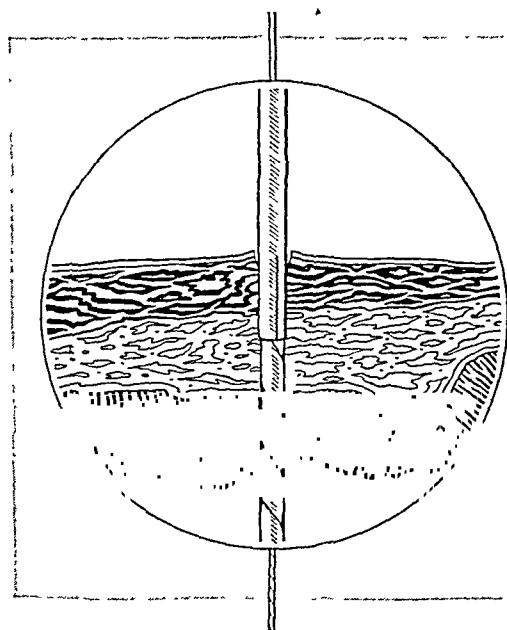
ATRAUMATIC NEEDLE—The needle and suture, as shown, have practically the same diameter—thus a minimum of tissue injury is produced

area to prevent turning in the needle holder.

Each special purpose suture has been developed in collaboration with recognized authorities and represents the consensus of professional opinion in its particular field.

In addition to the long list of Atraumatic suture-and-needle combinations specially designed for specific procedures, the D & G line presents a comprehensive range of non-needled sutures. These include catgut, kangaroo tendons and bands, ribbon gut, Anacap silk, Kal-dermic and Dermalon skin and tension sutures, and the various other non-absorbable materials.

D & G Sutures with Atraumatic Needles

INTESTINAL	THYROID	TONSIL	EYE	HARELIP
CLEFT PALATE	PLASTIC	NERVE	ARTERY	OBSTETRICAL
	CIRCUMCISION	URETERAL	RENAL	

Description and prices on request

Safe · Convenient

SAFER than non-boilable catgut because heat-sterilized *after* the tubes are sealed . . . tubes may be boiled or autoclaved to assure absolute asepsis of their outer surfaces.

MORE CONVENIENT because adaptable to any operating room technic . . . quickly prepared for use, as required, along with instruments or dressings . . . flexibility readily controllable to any degree.

These advantages, always important, assume special significance today when surgical personnel and facilities are under extraordinary pressure.

D & G Claustro Thermal Boilable Catgut

The American Journal of Surgery, Inc.

Reg. U. S. Pat. Off., Nov. 3, 1936

NEW SERIES VOL. LIX — Contents · March, 1943 — NUMBER THREE

The American Journal of Surgery

　　　　　　Contents · March, 1943　　　　　　

Case Reports

General Information

THE AMERICAN JOURNAL OF SURGERY does not hold itself responsible for any statements made or opinions expressed by any contributor in any article published in its columns.

Articles are accepted for publication with the understanding that they are original contributions never previously published. All manuscripts are subject to editorial modification, and upon acceptance become the property of THE AMERICAN JOURNAL OF SURGERY, INC.

A reasonable number of illustrations are supplied free of cost; special arrangements must be made with the editor and publishers for excess illustrations and elaborate tables.

Reprints are furnished on order. Prices are quoted when articles are in page form.

Material published in THE AMERICAN JOURNAL OF SURGERY is copyrighted and may not be reproduced without permission of the publishers.

Change of address must reach us by the 15th of the month preceding month of issue.

PREPARATION OF MANUSCRIPTS

Text. Manuscripts are to be typewritten, on one side of the paper, with double spacing and good margins. The original should be sent to the editor and a carbon copy retained by the author.

Illustrations. Illustrations must be in the form of glossy prints or drawings in black ink (*never* in blue). On the back of each illustration the figure number, author's name and an indication of the top of the picture should be given. Legends for illustrations are to be typewritten in a single list, with numbers corresponding to those on the photographs and drawings. Please do not attach legends to the pictures themselves.

Bibliographies. Bibliographic references should be at the end of the manuscript, in alphabetical order, and not in footnotes. Each reference should include the following information in the order indicated: Name of author with initials; title of article; name of periodical; volume, page and year. The following may be used as a model:

PHANEUF, LOUIS E. Indications and technique. *Am. J. Surg.*, 25: 446, 1937.

The author should always place his full address on his manuscript.

The subscription price of THE AMERICAN JOURNAL OF SURGERY, is $10.00 per year in advance in the United States; $15.00 in Canada; and $12.00 in foreign countries. Current single numbers $2.00. All Special Numbers $4.00. Prices for such back numbers as are available will be quoted on request.

Address all correspondence to

The American Journal of Surgery, Inc.

49 WEST 45TH STREET · NEW YORK

A PEACETIME DEVELOPMENT—
ASSIGNED A MAJOR ROLE IN WARTIME

WHEN introduced a few years ago, the G-E Photo-Roentgen Unit was acclaimed a most practical solution to the problem of conducting mass x-ray chest surveys economically—*yet efficiently.* For this unit, by the method of direct photography of the fluoroscopic image, made it feasible to use 4 by 5-inch instead of 14 by 17-inch films—at about one-tenth the material cost!

That hospitals, tuberculosis associations, city, county, and state health departments had long awaited this development, was soon evident, for today G-E Photo-Roentgen Units are fulfilling important assignments in all sections of the country.

And, as though this development had been timed for war-time service, G-E Photo-Roentgen Units were installed in scores of induction centers shortly after the Selective Service Act became effective. Thus

the U. S. Army Medical Department was quickly provided with a practical method for obtaining reliable radiographic records of millions of men—upon entering, likewise upon their discharge from service.

Interesting are the predictions of how medical science will further extend the use of these facilities in the peacetime to come, with results ultimately benefitting all walks of life.

Thus the considerable time and effort which a development of such far-reaching possibilities involves, again has proved fully justified.

GENERAL ⊕ ELECTRIC
X-RAY CORPORATION

Today's Best Buy – U. S. War Bonds

UPJOHN FERRATED LIVER CONCENTRATE

High Dosage IRON *Reinforced with Liver and B Complex Factors*

Ferrous Sulfate
Liver Concentrate
Vitamin B₁
Vitamin G
Nicotinamide
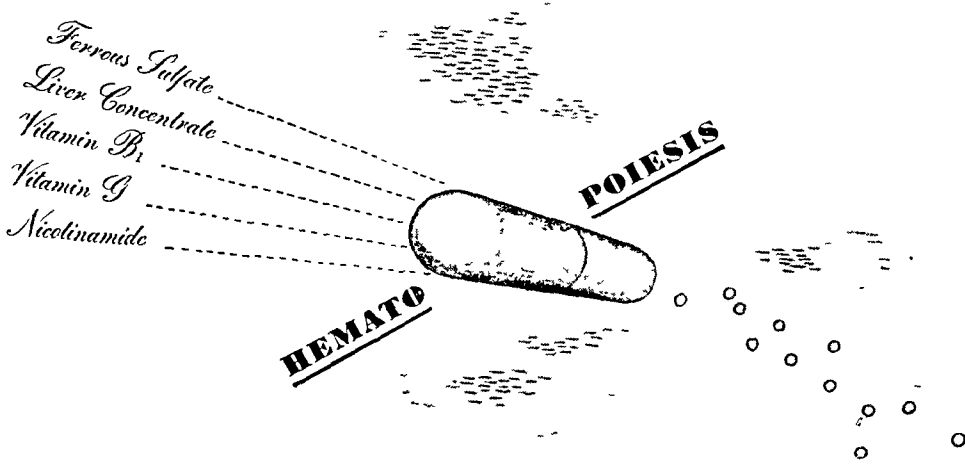
HEMATO

POIESIS

For the striking results possible in hypochromic ANEMIAS

of B complex deficiency, pregnancy, lactation and some

infections, Upjohn Ferrated Liver Concentrate

helps provide key factors essential to hematopoiesis.

Each capsule of Upjohn Ferrated Liver Concentrate
contains 3 3/5 grains Ferrous Sulfate; 7 grains
Liver Concentrate supplemented to present approx-
imately: 125 I. U. Vitamin B₁, 50 S. U. Vitamin G,
and 5 milligrams Nicotinamide.

Patients need only six capsules daily of Upjohn
Ferrated Liver Concentrate for potent, convenient,
economical therapy of hypochromic anemias.

Upjohn Ferrated Liver Concentrate

Available in bottles of 100 and 500 capsules

NOT
STRENGTH
ALONE

BUT ST NGTH

UNDER CONTROL

Not strength alone—rugged, unrestrained, variable—

but strength that is accurate, uniform, dependable. Of such is the

strength of Ethicon—assured by single-ownership production,

laboratory-supervised, from raw gut to finished strand. Ethicon is

exceptionally smooth, pliant, and, of course, conforms to U.S.P. standards.
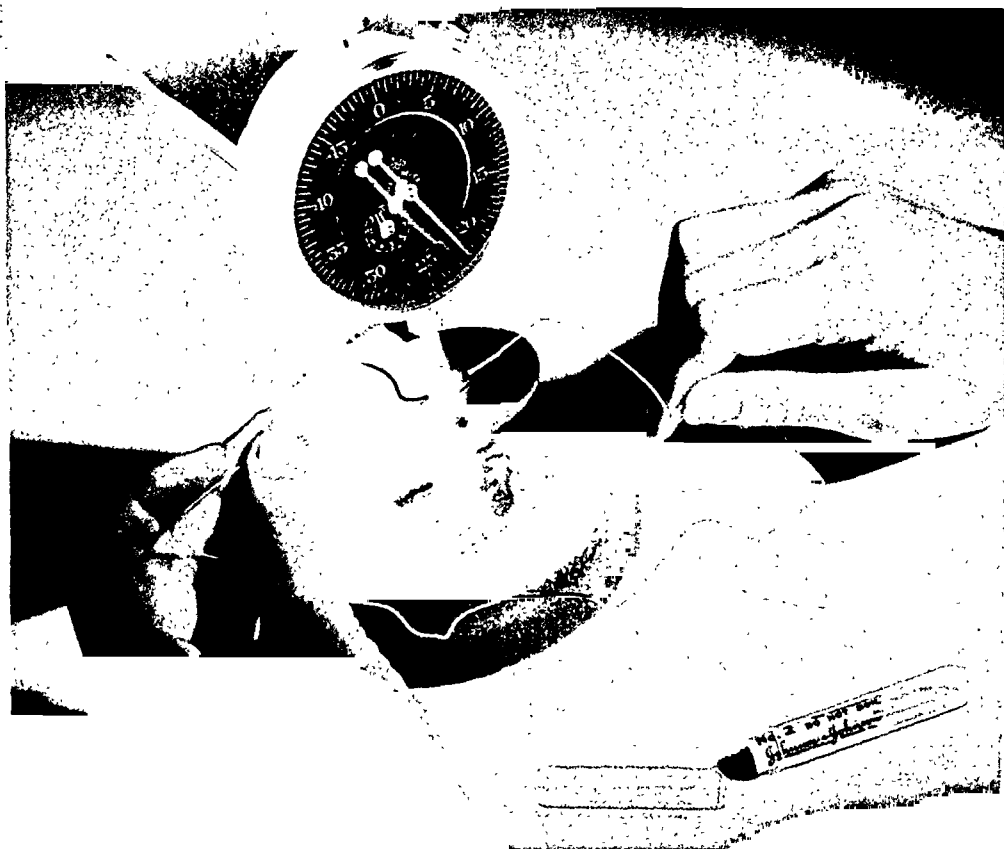
Not Strength Alone . . . But Uniformity of Strength. Ethicon Sutures combine maximum strength and minimum bulk, assuring the surgeon a suture that will hold tissue securely, yet involve the least possible amount of foreign substance in the tissue. Laboratory control of every process in the making of Ethicon results in sutures that are uniform in size and strength, worthy of the confidence of the surgeon who insists on the highest standards of material as well as of operating technique.

ETHICON SUTURE DIVISION

Johnson&Johnson

NEW BRUNSWICK, N. J.   CHICAGO, ILL.

SUTURE   MANUFACTURERS   SINCE   1887

Editorial

THE CANCER SPECIALIST OR ONCOLOGIST

DURING the past three or four decades the complexion of death-dealing diseases has passed through important changes. As everyone knows the improvement in general public hygiene and the control of communicable disease have greatly reduced the death rate under twenty-five years. In other words, a lesser number of people are dying in the first half of life. To this may be added the advances in diagnostic methods and medical and surgical therapeutics. Sociologists tell us that in the United States the control of emigration and birth control are other factors tending to make this country a country of old people. Whereas at the turn of the century the average duration of life in the United States was thirty-five, it is now close to sixty years. All of this means that the problem of controlling diseases has entered a new phase. Most of the deaths that occur today are from diseases developing after middle life. Foremost among these are cardiovascular diseases and second is cancer. These facts force the medical profession to devote more attention to the diagnosis and treatment of these death-dealing scourges.

We hear much nowadays—and rightly so—about the control of infantile paralysis. This is needed, for every year this dreaded disease leaves thousands of children crippled for life. It is astonishing, however, that cancer causes more deaths in childhood than does infantile paralysis.*

Another well recognized fact about medical progress is that medicine and surgery are rapidly becoming much more complicated arts and sciences than in the previous generation. Our increasing knowledge of the human body, its structures, functions and ills, requires a much more complicated system of diagnostic aids and gadgets and therapeutic weapons. All of this progress necessitates a much longer preparation for the practice of medicine and surgery, with emphasis upon specialization after a broad general foundation. Along with this increase in the complications of medicine, the diagnosis and treatment of cancer has likewise advanced. In bygone days, internal cancer was always fatal. The treatment of external cancer was a crude surgical or cautery procedure which perchance ended fairly happily if the disease was taken early. Countless numbers died of internal cancer without proper diagnosis. In recent years, surgery has made tremendous advances. Tumors in the brain, lungs, abdomen are now more frequently diagnosed and treated satisfactorily in the early stages. Operations for these serious conditions "have been made rela-

* Read, Clifton R. Annual Report of the American Society for the Control of Cancer, 22: 9, December, 1940.

tively safe for the patient and the patient is being made safe for the operation." The cautery, always a prominent part of the surgeon's armamentarium from time immemorial, is in many places being replaced by the refined apparatuses and technic of electrosurgery. The advent of radium and x-ray therapy are great boons to the cancer sufferer, not only in increasing the percentage of five-year salvages, but also in relieving the advanced cases. He who attempts to treat cancer today must have cognizance of all these therapeutic agents. One of my seniors once said, "Cancer is first to be treated by surgery and if this fails, then by x-ray or radium." This man lived to change this philosophy radically. Today each patient with cancer must be studied as an individual problem and the therapeutic agent or agents chosen which are best suited for him. To be able to make a careful selection of one or a combination of more than one of these therapeutic agents requires long and complicated training. Gradually a group of "specialists" have grown up who are cognizant of the intricate problems of cancer diagnosis and the elaborate methods of treatment.

The general trend of specialization must be carefully balanced by fundamental, comprehensive training. Too narrow limitations lead to many pitfalls through short-sightedness. On the other hand, it is impossible for any one man to master all fields. A happy medium must be worked out with time and experience. This is especially true in the realm of cancer therapy; no one man can be proficient in the diagnosis and treatment of all neoplasms widely distributed throughout the body. A prominent surgeon put it bluntly by saying, "No one can remove every kind of a tumor from the brain to the toenails!" Only the foolish would attempt such an inclusive "specialty." However, I can conceive of a type of specialist who is sufficiently well versed in the fundamental principles of neoplastic growth, its diagnosis and treatment, to be helpful in any clinic or large institution in solving many of the therapeutic problems.

He need not be a combination of brain surgeon, thoracic surgeon, gastrointestinal surgeon, urologist, gynecologist and radiologist. If, on the other hand, his training has been sufficiently broad by way of general medicine, general surgery, pathology and radiotherapeutics, he can be of value as a consultant in these fields and limit his own therapeutic efforts to a well defined and established specialty. For example: A well trained general surgeon who has studied pathology thoroughly and has had a year or two in radiotherapeutics, can continue as a general surgeon with special emphasis upon the treatment of neoplasms, develop skilled technic in his own field and be of inestimable value in co-operating with his confreres in other domains.

In recent years medical and surgical investigators have delved into physiological problems with far reaching discoveries. Cancer research has followed suit, increasing our knowledge of the behavior of cell growth and its relation to the cancer problem. The influence of hormones on cancer growth in the laboratory animal and patient has been studied with benefit. Just where this will lead is speculative. However, it means that the cancer therapist must keep abreast of these developments and be able to co-ordinate the results of laboratory investigation with clinical findings and make clinical applications of new discoveries when pertinent.

Although the "oncologist" may not be temperamentally able to or by training capable of carrying on highly technical laboratory, biological and physiological experiments, yet by keeping himself familiar with the advances in scientific research he can assist in correlating the work of the investigator and the clinician. In this manner a great service will be rendered the group or institution with whom he works. It is obvious then that in the larger institutions at least the whole cancer problem cannot be settled by one man alone, but the cancer specialist will be on hand to co-operate with a group of workers, including

the laboratory investigator, the pathologist, surgical specialists, readiologists, and at times the internist.

In smaller communities, the cancer specialist perhaps has a greater place to fill. He may be the only one in a small city serving a large surrounding territory, who has had besides his general medical foundation, training in pathology, surgery and radiation therapeutics. It is also important that in these communities, there be a well trained physician who can bring together all efforts, professional and civic, in establishing a program of cancer control, including education of the profession and lay public alike, and furnishing diagnostic facilities and therapeutic opportunities.

After this general discussion, it seems appropriate to make a few suggestions for the education of such persons. I will outline the training for a general surgical oncologist, bearing in mind that in larger cancer hospitals and other institutions, one or more individuals in each department may become especially qualified to manage the patients with cancer in the gynecological, urological or other departments.

In several hospitals in this country departmental cancer clinics have already been established and directed by a well trained physician.

Suggestions for General Surgical Oncologist. (The term "oncologist" is herein used for lack of a better one. It also obviates the somewhat distasteful title of "cancer specialist.")

First year, general internship, including medicine and surgical specialties.

Second year, pathology. Pathological training could be limited to six months, with the other six months devoted to a clinical specialty, thus increasing the general internship to eighteen months.

Third, fourth and fifth years—surgical internship, assistant residency and residency, respectively. Three years in surgery prepares the trainee for the American Board of Surgery, if he wishes to certify in that Board.

Sixth and seventh year—radiation therapeutics. This, of course, should include radium and x-ray therapy. With two years in radiation therapeutics and a foundation in pathology, the trainee would be eligible to apply for certification in the American Board of Radiology in radiation therapeutics.

Inasmuch as there is no Board of Oncology, an attempt has been made in the above outline for the trainee to emphasize either surgery or radiation therapeutics, so that he may certify in one of the established Boards. He cannot certify in more than one Board and therefore must choose his preference. It remains to be seen, as time goes on, and experience accumulates, whether an American Board of Oncology is really advisable.

A criticism of the above scheme might be raised as to the length of the training period. However, if one is to understand the cancer problem thoroughly and prepare himself to treat cancer patients in some one of the accepted specialties, six or seven years are certainly not too many.

In recapitulation then, one might say that the cancer specialist, or oncologist, is a practitioner of medicine, who has first of all a well founded knowledge of medicine. Following this he should have sufficient training in pathology, surgery and radiation therapeutics to understand the fundamental problems in cancer diagnosis and therapy. He cannot be expected to master all procedures for the treatment of all neoplasms wherever they may occur in the body. However, after a wide general cancer training he can specialize in some field to his liking and at the same time be of valuable consultatory aid to his confreres. In large general institutions, he can head up the group which operates the cancer clinic. In specialized cancer hospitals, he naturally would fall into a narrower field of one of the already accepted specialties. In smaller communities, as in a general hospital, he can be the leader in the cancer program.

GRANT E. WARD, M.D.

Original Articles

TRENDS IN SPINAL ANESTHESIA*

HENRY S. RUTH, M.D.   AND   D. DWIGHT GROVE, M.D.

Professor and Head of the Section of Anesthesiology, Hahnemann Medical College and Hospital of Philadelphia

Fellow in Anesthesiology, Hahnemann Medical College and Hospital of Philadelphia

PHILADELPHIA, PENNSYLVANIA

THE development of spinal anesthesia had its inception in a series of uncorrelated discoveries. From the time of the ancients, particularly Hippocrates and Galen, a few anatomical facts were known concerning the cerebrospinal nervous system. Cotugno discovered the presence of the cerebrospinal fluid in 1764.[1] Magendie was the first to show adequately that a section of the anterior roots of the spinal cord affected motility but not sensation, and the reverse was produced by severing the posterior roots. Thus he completed the work of Bell who had merely observed that the posterior roots could be cut "without convulsing the muscles," and a mere touch of the anterior roots caused convulsions.[1] This so-called Bell-Magendie law was confirmed in 1831 by Johannes Müller.[1] The cerebrospinal circulation was investigated and described by Magendie in 1842.[2] In 1851, Claude Bernard observed the results of cutting the sympathetic fibers of the nervous system.[1] In 1853, Pravaz published his original description of the first hypodermic syringe, which was improved shortly afterward by Alexander Wood in the same year.[3]

Cocaine, the active principle of erythroxylin coca, was known since ancient times by the South American natives under the name of the "divine plant of the Incas." Dr. Roy L. Moodie has reconstructed an artist's conception of an early surgical operation among the Incas, showing a blanket-clad native using the cautery to make a cruciform incision in the scalp of a woman suffering from melancholia. The operator chewed a cud of coca leaves, the juice from which would be dropped upon the wound if the pain became severe.[4] "Schezer obtained some of these leaves when on his scientific voyage around the worlds in the Austrian frigate, Novar, and observed that the leaves numbed the tongue when chewed. Gadecke, in 1855, isolated the alkaloid of the leaves and named it 'erythroxylin.' Niemann, in 1860,[5] obtained the alkaloid from them in a pure form, which he named 'cocain,' and also reported the numbing effect of this drug on the tongue; but no attention was paid to his discoveries. Indeed, Bennett, in 1874, demonstrated anesthetic properties of the drug without exciting notice, and four years later, van Anrep, after making a thorough investigation of the drug, could not arouse any interest in it, although he injected it hypodermically into himself. After noting that anesthesia lasted thirty-five minutes, he suggested that it might be used as an anesthetic for surgical purposes. In fact, in 1880, a British medical commission investigating this drug reported that it had no medical

value and was a poor substitute for caffeine."[6] It was not until 1884 that Carl Koller, an ophthalmologist, introduced cocaine as a local anesthetic agent and demonstrated its qualities in the eye.[5]

Dr. W. S. Halsted[7] in the winter of 1884–1885, freed the components of the brachial plexus imbedded in a cicatricial mass after injection of the roots of the plexus in the neck with cocaine. This operation was performed in a large tent on the grounds of Bellevue Hospital, "having found it impossible to carry out antiseptic precautions in the general amphitheater at Bellevue where numerous anti-Lister surgeons dominated and predominated." Halsted has received much of the credit as the prime mover of the cocaine investigation, initiating his contributions within a week after receiving the publication of Koller's first paper on cocaine, and which appear to have definitely established the principle of nerve blocking by regional anesthesia.

In the fall of 1885, Corning[8] published his first work on the action of cocaine on the spinal cord. As a neurologist, he was primarily interested in local medication of the cord and medical relief of pain, and not in the production of surgical anesthesia. Quoting Corning's original article, "I decided to inject the anesthetic between the spinous processes of the lower dorsal vertebrae. I was led to resort to this expedient from the knowledge of the fact that in the human subject numerous small veins run down between the spinous processes of the vertebrae and entering the spinal canal join the more considerable vessels of the plexus spinalis interna. From these theoretical considerations I reasoned that it was highly probable that, if the anesthetic was placed between the spinous processes of the vertebrae, it (the anesthetic) would be rapidly absorbed by the minute ramifications of the veins referred to, and, being transported by the blood to the substance of the cord, would give rise to anesthesia of the sensory and perhaps of the motor tracts of the same.

. . . I therefore anticipated a more or less local action of the drug upon the cord." He then injected 20 minims of a 2 per cent solution of the hydrochlorate of cocaine into the space situated between the spinous processes of two of the inferior vertebrae of a young dog. Five minutes later, he noticed inco-ordination and weakness of the hind legs and, subsequently, decreased sensitivity which persisted for a considerable length of time. His second reported experiment was the injection of 30 minims of a 3 per cent solution of the hydrochlorate of cocaine into the space between the spinous processes of the eleventh and twelfth dorsal vertebrae of a patient. After a lapse of six to eight minutes without symptoms he again injected 30 minims of the solution at the same spot and in the same manner. In about ten minutes the patient complained that his legs "felt sleepy," after which Corning confirmed the impairment of sensibility in the lower extremities of the lumbar region, the penis and the scrotum. He followed this by the painless passage of a sound which, on former occasions, had produced considerable distress. He concludes, "Whether the method will ever find an application as a substitute for etherization in genito-urinary or other branches of surgery, further experience alone can show. Be the destiny of the observation what it may, it has seemed to me, on the whole, worth recording." Corning definitely does not mention spinal puncture in his report and whether he accidentally punctured the dura or whether his results were obtained through an epidural injection may never be factually proved.

In Corning's second report,[9] it is evident that the injection of cocaine within the dura was not proposed by him at this time, for he elaborately describes an apparatus by which he made certain, by measurement of the distance from the skin to the posterior surface of the transverse processes, that the "foramen vertebrae" would not be entered during his injections. He

experimented with this principle of medication as a potent means of treating certain derangements of the spinal cord. The results cited on four human patients adequately demonstrate that he did not produce spinal anesthesia during the experiments alluded to in this second report. Prophetically enough, however, he concludes by trusting that his "long discourse may prove not without practical results."

As early as Corning's time there appears to have been some knowledge of the toxicity of cocaine, for in Corning's second series of experiments, he decreased the concentration of the injected drug from 3 per cent to 1½ per cent. Although of great significance, Corning's procedures remained little more than physiological experiments until a later period. In the meantime, in 1891, Quincke[10] demonstrated the feasibility of spinal puncture as a diagnostic procedure, but did not suggest its application to spinal anesthesia. As a result of the search of a drug possessing less toxicity than cocaine, Giesel isolated tropacocaine from the leaves of the Java coca plant.[5] The alkaloid of this drug was synthetically prepared by Lieberman in 1892.[11] Also, in 1897, beta eucain was discovered. Ziemssen[12] appeared to be the first individual to propose the use of local anesthetics by means of lumbar puncture. According to Dogliotti,[13] Corning, in 1894, advocated in his book[14] the insertion of a large needle into the intervertebral space, then the insertion of a finer needle through the first, and puncturing the subarachnoid space. Here, then, it would seem, rests the origin of the so-called fascia needle or "thumbtack" rather widely used today. Sicard,[15] in 1898 and 1899, experimented with many substances injected by means of lumbar puncture. These works led up to the real event of August Bier,[16] of Kiel, Germany.

This famous surgeon, on August 16, 1898, found it necessary "to amputate a foot in a tuberculous patient who had already undergone several procedures under anesthesia. Bier conceived the idea of injecting 3 cc. of a 0.5 per cent solution of cocaine into the dural sac. The result was startling, the patient having complete anesthesia of both lower limbs for over an hour, followed by some vomiting and a mild headache for twenty-four hours. The convalescence was complete, to the justified gratification of Bier and his assistants.

"After having used spinal anesthesia successfully in six cases without any complications, Bier decided to have a spinal anesthesia tried on himself. He had his assistant, Hildebrandt, inject 2 cc. of a 1 per cent solution of cocaine into the dural sac. Hildebrandt, not to be outdone by his chief, had the same done to himself. The anesthesia was highly satisfactory, but the intolerance of the two celebrated surgeons was much more than that of their patients. They were confined to bed nine and four days, respectively, with vomiting, severe headaches, and marked vertigo. After this period of time they returned to normal, but their personal experience by this method was of infinite value. Bier gave a marvelous description of spinal anesthesia, bringing out its good points and at the same time its defects."[13] After these careful observations, Bier himself asserted that the spinal route should not be acceptable for the production of spinal anesthesia until some substitute for cocaine free from its disagreeable by-effects could be found.[17]

Tuffier of France,[18] however, took up the torch and became the most pronounced advocate of the method at this period. He gave brilliant demonstrations before the crowded audience that came from all parts of the world to attend the Thirteenth International Medical Congress held in Paris during the summer of 1900.[7] It was Tuffier's work that aroused the interest of American surgeons.

Little attention has been given in the literature to the first application of spinal anesthesia in America, due to the fact

that the surgeons, Tait and Caglieri of San Francisco,[19] who initially administered it, were not the first to report their work in the literature. On October 26, 1899, they performed an osteotomy of the tibia under spinal anesthesia, using 1 cc. of a 0.75 per cent solution of cocaine. The patient was slightly passed middle age and the operation was apparently successful, without pain or discomfort. An analogy may be drawn between the application of inhalation and spinal anesthesia in this country in that the first report of each was recorded by persons other than the ones who actually performed the initial application.

The first report of the administration of spinal anesthesia for surgery in America appeared in the Journal of the American Medical Association under New Orleans in Medical News.[20] Under the caption of "Spinal Cocainization," this report reads, "The Bier method of intraspinal cocainization was applied successfully in the Charity Hospital, here, on December 18, by Dr. Matas, assisted by Drs. F. A. Larue and H. B. Gessner, and Mr. Allen, interne. The patient, an Argentine *mestizzo* of 32 years, suffering from internal hemorrhoids, ulcerated, bleeding and excessively tender, had albumin and casts in the urine. A needle having been introduced in the median line, between the fifth lumbar vertebra and the sacrum, the entrance of the needle into the canal was readily confirmed by the escape of cerebrospinal fluid. Two doses of 1 per cent normal salt solution of cocaine hydrochlorate, each of 1 cc., were injected five minutes apart. Nineteen minutes after the first, an internal hemorrhoid was clamped, a groove made around it with scissors, and a ligature applied, and, following this, the distal portion was snipped off; all of this absolutely without any pain. Anesthesia of the lower extremities, the penis, scrotum, and perineum, the abdominal wall, the chest and, in fact, the entire body below the neck, lessening in a degree, however, from below up, was subsequently observed.

The procedure was followed by the usual chill, fever to 103.4°F., nausea, occipital headache. Prof. Matas had previously— November 10—applied the method in the case of a colored man, aged 23 years, suffering from a tuberculous knee-joint: 1 cc. of a solution of eucain B, in two doses, was injected through a needle between the first and second lumbar vertebrae. Although the entrance into the canal was confirmed by the escape of cerebrospinal fluid, followed by the usual systemic reaction, no anesthesia was produced, the quantity of eucain being perhaps insufficient." An interesting commentary may be made in that the interne, Dr. Carroll W. Allen, who assisted Dr. Matas, subsequently contributed a textbook on local and regional anesthesia.[21]

In his very complete article in the Philadelphia Medical Journal,[22] Doctor Matas states, "The simple fact that so many experienced surgeons the world over have accepted the new and yet unproved methods of spinal cocainization as a substitute for the general anesthetic is the best proof of dissatisfaction in existing conditions, and is a practical admission of the fact that there is still much room for improvement. The danger now lies in the other direction. In our efforts to escape from the dangers of Charybdis, we are running into the perils of Scylla; and it is not unlikely that we shall witness a repetition of the same errors which so disastrously characterized the early history of cocaine anesthesia and which threatened for a time to bar it out of the field of general surgical practice. The wave of enthusiasm which is at present sweeping over this country will undoubtedly furnish the necessary data needed to form a true estimate of the possibilities and the limitations of the method. But at what cost we cannot tell. In the meantime, the general anesthetics will remain as they must ever with us; if not in the present form, in some other and in spite of their dangers and disadvantages, because unconsciousness in surgical opera-

tions is still a desideratum in many interventions if only to abolish the psychic pain which at times cannot be prevented by the most complete analgesia. At this moment a wave of curiosity blended with enthusiasm is sweeping over the entire country, and almost every surgeon is impatient to try or he has tried to report the 'new method.' It is not likely that communications bearing upon less brilliant methods which require time and much patience to acquire will be received with special favor."

Matas had the foresight to condemn both the injections into the dorsal and *cervical portions of the spinal canal and the* use of larger doses than those prescribed by Bier and Tuffier. Again quoting Matas, "Special attention must be given to the exact dose of cocaine injected and to the aseptic preparation of the solution. Bier, in the first cases operated from August 16, 1898, to April, 1899, used 2 cc. of a 1 per cent solution. He never exceeded 15 mg. Tuffier, whose experience with the method is greater than that of any other operator (130 cases up to September, 1900), invariably uses a 2 per cent solution of cocaine, of which he injects 1 cc. The total dose, he insists very correctly, should never exceed 15 mg. In my own practice I began by using 1 cc. of a 1 per cent solution of eucain B. after the flow of cerebrospinal fluid had been clearly demonstrated; the needle, with the syringe attached, was left in situ pending observation of the anesthetic phenomena; after ten minutes' delay, as there was no anesthetic effect, another cubic centimeter was injected, and the cannula was withdrawn. Absolutely no effect followed. It would appear from this experience that eucain B. in the same doses as cocaine is absolutely inefficient." Matas further states that care must be exercised to avoid any unnecessary loss of cerebrospinal fluid, as serious accidents had followed excessive drainage in several cases. Bier himself was made quite ill from this cause when the method was tried on himself. At this period in the development

of spinal anesthesia, the main interest centered upon the proper method of accomplishing spinal puncture, at what location it should be performed and upon the toxicity of cocaine.

About this time, another factor mitigated against the continued popularity of spinal anesthesia. Corning,[23] Kummer, and others discovered that cocaine anesthesia can be indefinitely prolonged by arresting circulation in the anesthetized area of the extremities by using circular elastic bandages.[5] Braun, who had already published his work on the toxicity of cocaine (1897),[5] in 1901 introduced the use of adrenalin,[24] *the chemical tourniquet, and the elastic* tourniquet was abandoned.[5] By rendering nerve block or conduction anesthesia, as it was then termed, safer than prior to the use of epinephrine, the popularity of peripheral nerve block increased over spinal anesthesia. Matas himself entered a most logical plea for the use of regional anesthesia over spinal because of the fatalities and complications encountered with spinal anesthesia. After careful observations, Bier himself asserted that the spinal route should not be acceptable for the production of spinal anesthesia until some substitute for cocaine free from its disagreeable by-effects could be found.[7] In a rather large collection of reports on spinal anesthesia in 1900 in the Philadelphia Medical Journal, many surgeons reported their enthusiasm for the use of spinal anesthesia, but every report included discouraging aspects. Due to these complications many surgeons lost all interest in spinal anesthesia. The wave of criticism was further increased by attempts to anesthetize the whole body under spinal anesthesia. An outstanding example of the radical nature with which spinal anesthesia was applied may be cited by the work of LeFilliâtre in the years 1902 to 1904.[24] With his technic, the patient was placed in the sitting position, and he withdrew from 10 to 30 cc. of cerebrospinal fluid, the amount depending on the height of anesthesia desired, the maximum quantity when anesthesia of the

head was to be instituted. Varying amounts of 1½ per cent cocaine were then injected by barbotage. By his method of barbotage, this intrepid surgeon made the injection, allowed the 3 cc. syringe to refill to its capacity, reinjected, and repeated the procedure three or four times. Others injected as high as the sixth cervical interspace. One does not marvel that such methods produced disastrous results which, together with the toxicity of cocaine, were responsible for the very marked decline in the use of spinal anesthesia in the years of 1903 and 1904. At this time the method apparently suffered its first period of real unpopularity.

Condemnation of the use of cocaine continued, and the search for less toxic agents was again stimulated. In 1904, Fourneau,[24] a Frenchman, synthesized a new compound which he termed stovaine after the English translation of his own name. The new drug was less toxic and more stable than cocaine and its advent re-awakened some interest in spinal anesthesia. Our former acquaintance, Tuffier, Reclus and other French surgeons received it with enthusiasm. Babcock,[25] of Philadelphia, studied spinal anesthesia in France and began using it in his surgical service in Philadelphia. Later, he contributed to its safety by emphasizing the value of mouth-to-mouth resuscitation. Another impetus was provided in 1905 when Einhorn,[26] a German, synthesized novocaine. Although this drug is the least toxic of spinal anesthetic agents in use at the present time, its comparative innocuous nature was not fully recognized for many years. After a few trials with novocaine, the pioneers of subarachnoid block returned to the stovaine. Some surgeons used tropacocaine.[11] In the same year, Dixon[27] published his studies on the selective activity of local anesthetics upon the sensory and motor nerve fibers, showing that the sensory components succumbed to their action more rapidly and in lower concentration than do the motor.

In spite of being provided by newer drugs of lesser toxicity, too little as yet was known of the mode of action of spinal anesthesia for it to regain all of its former, original popularity. Sudden extensions of anesthesia after injection and unexplained fatalities were reported. A few, however, were still not disturbed. Barker,[28] an Englishman, in 1907, attempted to control the level of anesthesia produced by spinal methods by a formula which used stovaine as the drug and increasing the specific gravity of the injected solution by means of 5 per cent glucose. Experimenting with such a solution he apparently was the first individual to recognize the importance of the normal spinal curves in their effect upon the ultimate location within the spinal column of the injected solution when dealing with hyperbaric or heavy solutions. Chaput[24] attempted similar results with stovaine, increasing the specific gravity of the solution with 10 per cent sodium chloride. His results, however, were not as successful. Jonnesco, a Roumanian surgeon, in 1908, believed that he had a new and novel method of producing anesthesia for various levels by shifting the level of the spinal puncture. He also advocated the addition of strychnine to the injected solution, believing it to be a stimulant to the spinal nerves. In 1910, he toured the United States in an effort to popularize the method. Unfortunately, he, at times, failed in accomplishing high spinal puncture and the instances of sudden respiratory collapse were too frequent.

During this second period of decreasing popularity of spinal anesthesia Babcock[24] advocated his hypobaric or lighter-than-spinal-fluid solution. This consisted of stovaine in distilled water, the strychnine first advocated by Jonnesco, lactic acid to maintain the stovaine in solution, and the addition of alcohol to reduce the specific gravity. He further believed that the alcohol was of value in aiding sterility of the solution.

Essentially, perhaps, the lack of knowledge of the fundamental principles of spinal anesthesia caused the second period of

minimal use of spinal anesthesia in this country from 1910 to 1920 in spite of the provision of safer agents and further experimentation already mentioned. The dangers of spinal anesthesia, poorly understood as it was at that time, were further emphasized by the comparative safety of the then more or less standardized open drop ether technic in spite of the remote dangers and complications of the latter method. Criticism of spinal anesthesia in this interval reached its peak. The late great John B. Murphy, who had originally hailed spinal anesthesia with some enthusiasm, was now so antagonistic to the method that he suggested that it be prohibited by law.[29] During this period, European surgeons employed spinal anesthesia to a greater extent than in this country due, perhaps, to the contrasting attitude shown on the continent toward free patients and also because inhalation anesthesia was not as well understood in those countries as here. The repercussions of the fear of spinal anesthesia generated during this decade are still encountered all too frequently today.

According to Blumfield and McCardie,[30] the death rate at that time was estimated at 1 to 800–1000, which was considerably higher than with inhalation. Headaches appeared in 33 to 50 per cent of instances, backache in 30 per cent and abducens paralysis was frequent. He concluded "that spinal analgesia is retrogressing in favor and is generally only used when there is marked contraindication to inhalation anesthesia and local anesthesia is not possible. In the treatment of emergencies from spinal analgesia one does wrong whether one sits up or inverts the patient—in the one case he may die of syncope and in the other of poisoning."

An additional commentary on the use of spinal anesthesia of the period has been expressed by Pitkin:[31] "When I started using spinal anesthesia in 1912 its use was confined to very elderly people, those considered as 'bad risks' and to whom we were afraid to give ether. In those days we knew little or nothing about blood pressure changes, and our only concern was whether we had anesthesia—we used to marvel at the fact that we had such nice dry wounds." Of late, the profession realizes that the spinal method is frequently the ideal anesthesia for the young robust individual and many times is contraindicated in the individual of poor physical status.

Labat,[32] too, states that "in 1920 the prejudice against spinal anesthesia was so great that grave concern was considered before spinal was given. Between the years of 1913 and 1921 interest in the subarachnoid block seemed to have been declining all over the world except in 1919 which was particularly rich in foreign publications. In the United States three articles only were published in 1921."

Labat[33] was largely responsible for a sudden increase in popularity of spinal anesthesia beginning about 1920 which has continued to the present time in only slight variations of enthusiasm. His teachings included three factors essentially, two of which had value. His first, the recommendation to employ neocaine, has been subjected to just criticism, as the drug proved to be nothing more than procaine in a French ampule. His two suggestions of value rested in the barbotage technic and the compulsory use of the Trendelenburg position. By barbotaging the solution of procaine crystals dissolved in spinal fluid, the specific gravity of the injected solution was so reduced that it approached isobaric levels. (By isobaric is meant a specific gravity approximately that of spinal fluid.) Subsequently, when he placed the patient in Trendelenburg position there was but little spread of the anesthetic solution cephalad. The Trendelenburg position had value in facilitating through gravity the blood supply to the cerebrum. Labat, in one statement[34] claims to have used the Trendelenburg position since 1916. Later[32] he states, "Our first demonstration in the United States of the use of Trendelenburg position after spinal injection of a simple solution of neocaine dissolved in the

cerebrospinal fluid was given on October 20th at the St. Mary's Hospital of the Mayo Clinic." Despite these discrepancies in time, Labat's teachings did much to establish a recrudescence of spinal anesthesia.

By 1924, increasing concern was being expressed over the fall in blood pressure initiated by spinal anesthesia. Little could be accomplished to combat it save the Trendelenburg position advocated by Labat. In 1924, the active principle of ma huang, ephedrine, was introduced by Chen.[35] Originally, its use was reported by him in experimental shock and hemorrhage. In 1927, Ockerblad and Dillon[36] suggested its use as a protective agent to combat the extreme blood pressure fall of spinal anesthesia. Again, a wave of overenthusiasm threatened the use of ephedrine in that excessive doses to the point of disorganization of cardiac conduction and fibrillation were employed by some. The reaction against the use of ephedrine was led by Labat who expressed the belief that through its action of vasoconstriction in the unanesthetized area the blood supply in that area was further decreased. As a result of experimentation and clinical application the use of vasoconstrictors is well established today, and additional drugs such as neosynephrine, paredrinol and others have been provided, although in general, with the exception of ephedrine, they may be considered as still being in the experimental stage.

In 1928, Pitkin[31] provided a new impetus to spinal anesthesia through his writings. Although his solution of procaine, which he termed "spinocain" is but little used today, and his technic in general is followed but rarely, his publications at the time were indeed well received, and served to focus further attention on subarachnoid block.

Since 1930, spinal anesthesia has enjoyed a period of stability in popularity, more or less confidence in its action and more logical application than prior to this period. This attitude has now been accomplished by the greater knowledge now possessed

concerning the many factors involved, which knowledge has been derived from both experimental and clinical data.

Some of the aspects which are better understood and consequently better managed, and some of the more recent outstanding contributions may be briefly epitomized as follows:

Selection of Patients. Certain patients do not do well under spinal anesthesia. It is not necessary nor particularly advantageous for certain surgical procedures. Complications such as severe anemias, shock, particularly from hemorrhage, and others should preclude its use. These factors are well understood, and usually are not ignored, to the advantage of the present popularity of the method.

Preanesthetic Sedation. Psychic reactions are controlled to a large extent by the individualized administration of drugs to the degree of sedation desired. Morphine is employed for its analgesic and hypnotic qualities, atropine or scopolamine because they possess the ability to depress certain reflexes, and the short acting barbiturates to increase the hypnosis. Through the preoperative use of the above drugs, the patient is rendered more comfortable and safe during the operative period, although excessive degrees of central depression should be avoided. No hesitation is shown in administering additional doses when indicated; ofttimes the intravenous route is employed for such supplementation.

Anesthetic Agents. Metycaine, pontocain and nupercaine are the more popular of the newer drugs. The pharmacological action of each is well understood, and due allowance should be and is usually made for their property of increased toxicity. Their outstanding contribution has been through greater duration of action as compared to procaine.

Technics. Standardization of uncomplicated technics has been well described by Lundy, Tovell and others. By these comparatively simple technics, control over the height of anesthesia is readily attained in most instances, and the anesthetization of

excessive and unnecessary areas avoided. Control of prolonged anesthesia of the upper part of the abdomen has been gained by the elaboration of the older technics employing solutions of lighter or heavier specific gravity than that of spinal fluid, through the contributions of Sise,[37] Jones,[38] Etherington-Wilson[39] and others. The effect of position and the presence of the normal spinal curves have also been well described.

Care of Patient during Anesthetic Period: Oxygenation. Mainly through the contribution of Waters and Seevers, the prevalence and the ill effects of oxygen want under spinal anesthesia have received emphasis. Decrease of oxygen content and increase in carbon dioxide content of the blood are present when blood pressure is low. Cellular oxygen want depresses the sensitivity of the vasoconstrictor center. The efficiency of ephedrine is likewise decreased under such circumstances. Inhalations of pure oxygen are, therefore, indicated and prescribed when falling blood pressure occurs. Anesthetists are ever on the alert for the well defined manifestations of oxygen want.

Maintenance of Blood Pressure. Vasoconstrictors are employed in a logical manner. Prevention of excessive drop in blood pressure is attempted by their prophylactic use. A fall in blood pressure is ignored when slight, but the blood pressure is constantly observed, and if the fall becomes excessive, it is rectified by additional administrations of these drugs in small but effective doses.

Combination with Other Agents and Methods. Before the present period of spinal anesthesia, rather firm reluctance was felt toward supplementation unless absolutely necessary, because of the absence of agents applicable for such purposes and the consequent dangers involved by their use. Through the provision of cyclopropane and pentothal, especially, supplementation is now facilitated and made safe. The use of these two agents many times in minute quantities easily control certain complications such as nausea and vomiting,

restlessness, tenseness and fear. Indeed, many clinicians intentionally and initially combine them with excellent results with respect to safety, efficiency and comfort, to both surgeon and patient.

Physiological Effects of Spinal Anesthesia. The large mass of experimental work completed on spinal anesthesia since 1928 has greatly enlarged our knowledge of its effects and derangements. Suffice it to say that such contributions have added largely to the safety of the method as employed today.

In conclusion, one advance in spinal anesthesia of recent origin has assumed such importance that it should not be ignored in a presentation of a historical and review nature. I am referring to the method termed "continuous," "serial" or "fractional" spinal. The method was devised by Lemmon[40,41] in order to be able to prolong the anesthetic period as long as it was necessary or expedient to do so. The method has particularly appealed to those anesthesiologists and surgeons who desired prolongation of anesthesia but were reluctant to employ drugs with greater duration of action because of their increased toxicity. With Lemmon's method, additional amounts of anesthetic drugs may be injected into the subdural space at will. To employ this technic a special needle is used which is made of a silver alloy which may be bent into sharp angles without breaking; tubing made of firm rubber with an inside diameter of 2 mm., about 30 inches in length, fitted with a Luer-lok connection to the spinal needle at one end and a stop-cock adapter to the standard Luer syringe at the other end; and a mattress which has an opening through its entire thickness which permits access to the needle *in situ* during the operation. The mattress also prevents pressure on the needle, and compression of or tension on the tubing by the body weight of the patient. The patient is placed on the mattress in the lateral position for the spinal puncture with the special needle. An appropriate quantity of the anesthetic agent in solution is placed in the syringe,

the tubing attached to the syringe, and filled with the solution to remove the air, and the stop-cock closed. One and one-half, 3, 5 and 10 per cent solutions have been employed, but the 3 and 5 per cent concentrations appear to be most popular. The tubing and syringe are finally attached to the spinal needle and the patient placed in position for the surgical procedure. The selected estimated initial dose is administered, after which additional amounts may be injected from time to time as the necessity arises.

The advantages[42] of this method of spinal anesthesia are derived from the factor of controllability and include a smaller initial dose, giving increased safety; anesthesia may be maintained over a prolonged period; failure may be prevented in the rare patient who requires a larger dosage; and an opportunity is presented for acquiring more knowledge of spinal anesthesia. The disadvantages encountered are largely technical in nature and occur with sufficient frequency to discourage its use when one is certain that a single injection is adequate. These disadvantages may be listed as: technical difficulty of introducing the malleable needle; the factor of slightly longer time required for its application; displacement of the needle may occur when the patient is moved; and there may be an increased possibility of trauma to the structures at the site of injection as compared with the conventional type of spinal anesthesia. The indications for this method will vary, but assuming one has determined to use procaine for spinal anesthesia, the general indications are: upper abdominal procedures requiring more than one hour and lower abdominal operations of greater length; exploratory laparotomies that may be either short or long in duration, depending upon the condition encountered; and patients in poor physical status—in order to avoid a large single injection.

Spinal anesthesia will no doubt assume an important rôle in the casualties of World War II. The present war differs from prior ones in that the aeroplane has been responsible for almost as large a casualty list among noncombatants as among the personnel of the armed forces in the active zones. Spinal anesthesia is impractical at Clearing Stations,[43] which are stations nearest the front where anesthesia is employed, because of the temporary nature of these hospitals. In Evacuation and Base Hospitals, spinal anesthesia is popular, unless the patients are severely shocked or in a hemorrhagic state, in which case experience in the last war made it clear how harmful the method can be in the shocked and hemorrhagic wounded.[44] Its compact nature and ease of continuity of supply account for its applicability here. If the wounded are further complicated by having been subjected to gassing, it appears that spinal and regional anesthesia should be used wherever possible as the method of choice, particularly when the patients are in the established stage, as compared to the initial stage.[45] For the treatment of naval personnel, in the sick bay itself, the risks of explosion and the lack of help narrow the choice of anesthesia to nitrous oxide, pentothal, regional and spinal anesthesia.[43] There is a possibility that the necessity of the widespread use of subarachnoid block during this appalling conflict may write another and most interesting chapter in the history of spinal anesthesia.

REFERENCES

1. Castiglioni, Arturo. A History of Medicine, New York, 1941. A. A. Knopf.
2. Magendie, F. Recherches Physiologiques et Cliniques sur le Liquide Céphalorachidien ou cérébro-spinal, Paris, 1842. Mequignon-Marvis.
3. Wood, Alexander. Quoted by Gwathmey, J. T. Anesthesia. Ed. 1, p. 478. New York, 1914. The Macmillan Co.
4. Leake, C. D. The historical development of surgical anesthesia. *Scien. Monthly*, 20: 304–328, 1925.
5. Koller, Carl. Über die Verwendung des Cocaïn zur Anäesthesirung am Auge. *Wien. med. Bl.*, 7: 1352, 1884.
6. Bumpus, H. C., Jr. History of regional anesthesia in urology. *J. A. M. A.*, 96: 83–88, 1931.
7. Matas, R. W. Local and regional anesthesia. *Am. J. Surg.*, 25: 189–198, 1934.
8. Corning, J. L. Spinal anesthesia and local medication of the cord. *New York State M. J.*, 42: 483–485, 1885.

9. CORNING, J. L. A further contribution on local medication of the spinal cord with cases. *Med. Rec.*, p. 291–293, 1888.

10. QUINCKE, H. Die Lumbalpunction des Hydrocephalus. *Klin. Wchnschr.*, 28: 929–975, 1891.

11. LABAT, GASTON: Regional Anesthesia; Its Technic and Clinical Application. Ed. 2. Philadelphia, 1928. W. B. Saunders Co.

12. ZIEMSSEN. Quoted by Tait, D., and Caglieri, G. Experimental and clinical notes on the subarachnoid space. Abst. *J. A. M. A.*, 35: 6–10, 1900.

13. DOGLIOTTI, A. M. Anesthesia: Narcosis, Local, Regional, Spinal, Chicago, 1939. S. B. Debour.

14. CORNING, J. L. Pain in a Dural Pathological, Prognostic, Medicolegal, and Neurotherapeutic Relation. Quoted by Allen, C. W.: Local and Regional Anesthesia. Ed. 2, p. 433. Philadelphia, 1918, W. B. Saunders Co.

15. SICARD. Quoted by Tait, D., and Caglieri, G. Experimental and clinical notes on the subarachnoid space. Abst. *J. A. M. A.*, 35 6–10, 1900.

16. BIER, AUGUST. Versuch über Cocainisirung des Rückenmarkes. *Deutsche Ztschr. f. Chir.*, 51: 361–369, 1899.

17. Editorial. *Am. J. Surg.*, 29: 33–41, 1910.

18. TUFFIER. Soc. de Chir. (Nov.) 1899; Tuffier and Hallion: Effets circulatoires des injections sousarachnoïdiennes de cocaïne dans la région lombaire. *Compt. rend. Soc. de biol.*, 52: 897, 1900.

19. TAIT, D. and CAGLIERI, G. Experimental and clinical notes on the subarachnoid space. Abst. *J. A. M. A.*, 35: 6–10, 1900.

20. MATAS, R. Report of successful spinal anesthesia. *J. A. M. A.*, 33: 1659, 1899.

21. ALLEN, C. W. Local and Regional Anesthesia. Ed. 2, p. 432. Philadelphia, 1918. W. B. Saunders Co.

22. MATAS, R. Local and regional anesthesia with cocain and other analgesic drugs, including the subarachnoid method, as applied in general surgical practice. *Philadelphia M. J.*, 6: 820–843, 1900.

23. CORNING, J. L. On the prolongation of the anaesthetic effects of the hydrochlorate of cocaine when subcutaneously injected; an experimental study. *New York M. J.*, p. 317–319, 1885.

24. MAXSON, L. H. Spinal Anesthesia. Philadelphia, 1938. J. B. Lippincott Co.

25. BABCOCK, W. W. Twenty-four years' experience with spinal anesthesia. *Am. J. Surg.*, 5: 571–575, 1928.

26. GOODMAN, L. and GILMAN, A. The Pharmacological Basis of Therapeutics, New York, 1941. The Macmillan Co.

27. DIXON, W. E. The selective action of cocain on nerve fibers. *J. Physiol.*, 32: 94, 1905.

28. BARKER, A. E. Clinical experiences with spinal anesthesia in 100 cases. *Brit. M. J.*, 1: 665–674, 1907.

29. MURPHY, J. B. General Surgery. Ed. note in *The Practical Medicine Series*, 2: 28, 1909.

30. BLUMFIELD, J. and McCARDIE, W. J. *Brit. M. J.*, p. 762–766, 1910.

31. PITKIN, G. P. Controllable spinal anesthesia. *J. M. Soc. New Jersey*, 24: 425–438, 1927; Controllable spinal anesthesia. *Am. J. Surg.*, 5: 537–553, 1928; Spinal anesthesia. *J. A. M. A.*, 91: 819–820, 1928; Controllable spinal anesthesia. *Brit. J. Anaesth.*, 6: 22, 1928; A nonoxidizing epinephrine to prolong spinal anesthesia with a subarachnoid capacity control. *Anesth. & Analg.*, 19: 241–260, 1940; 19: 315–329, 1940. PITKIN, G. P. and McCORMACK, F. C. Controllable spinal anesthesia in obstetrics. *Surg., Gynec. & Obst.*, 47: 713–726, 1928.

32. LABAT, GASTON. The trend of subarachnoid block. *Surg. Clin. North America*, 10: 671–681, 1930.

33. LABAT, GASTON. Local, regional and spinal anesthesia. *Ann. Surg.*, 74: 673–683, 1921; Regional Anesthesia, Its Technic and Clinical Application, Philadelphia, 1922. W. B. Saunders Co.

34. LABAT, GASTON. Correspondence. *J. A. M. A.*, 91: 514, 1928.

35. CHEN, K. K. and SCHMIDT, C. F. *J. Pharmacol. & Exper. Therap.*, 24: 339, 1924.

36. OCKERBLAD, N. F. and DILLON, T. G. The use of ephedrine in spinal anesthesia; preliminary report. *J. A. M. A.*, 88: 1135–1136, 1927.

37. SISE, L. F. Pontocaine and glucose solution for spinal anesthesia. *Surg. Clin. North America*, 15: 1501–1511, 1935.

38. JONES, W. H. Spinal analgesia—a new method and a new drug—percaine. *Brit. J. Anaesth.*, 7: 99–113, 1930; 7: 146–156, 1930.

39. ETHERINGTON-WILSON, W. Intra-thecal nerve root block. *Brit. J. Anaesth.*, 11: 43–55, 1934.

40. LEMMON, W. T. A method for continuous spinal anesthesia. A preliminary report. *Ann. Surg.*, 111: 141–144, 1940.

41. LEMMON, W. T. and PASCHAL, G. W., JR. Continuous spinal anesthesia. *Pennsylvania M. J.*, 44: 975–981, 1941.

42. HAUGEN, F. P., RUTH, H. S. and TAYLOR, I. B. Serial spinal anesthesia. *Anesthesiology*, 3: 52–60. 1942.

43. METCALFE, R. F. and WILLCUTS, M. D. Practical procedures for anesthesia and analgesia in war surgery. *Internat. Congr. Mil. Med. & Pharm.*, 10: 183–195, 1939.

44. MALLINSON, F. B. Casualty anaesthesia in the E. M, S. *Brit. J. Anaesth.*, 17: 98–107, 1941.

45. MONOD, R. and HAMBOURGER, P. L'anestesie des blessés gazés. *Presse méd.*, 47: 1437–1441, 1939.

THE MANAGEMENT OF THE RETAINED CERVICAL STUMP*

A BRIEF ANALYSIS OF FORTY-FOUR CASES

CURTIS H. TYRONE, M.D.   AND   JOHN C. WEED, M.D.†

Diplomate of the American Board of Obstetrics and
Gynecology

NEW ORLEANS, LOUISIANA

THE frequency of the preformance of hysterectomy for the treatment of uterine diseases has created a problem that is presented too often to a busy gynecologist. This problem is the management of the cervical stump. Although total hysterectomy is becoming more and more widely performed at the present time, Kennedy[2] estimates that 95 per cent of all the hysterectomies performed in the country are subtotal. Larger clinics[4,6,7] have inclined toward the total operation, but the general surgeon and the occasional operator have not followed this trend. Perhaps the chief reason for this is that the total operation is technically more difficult in untrained hands. Another possible cause is the fact that diseases of the cervix are often discounted or overlooked when hysterectomy is contemplated.

That a diseased cervical stump can and does give rise to distressing symptoms has received but little attention in medical literature. In a series of 6,550 subtotal hysterectomies performed at the Johns Hopkins Hospital during a forty-two-year period, Henriksen[5] reported that 131 women (2 per cent) returned complaining of symptoms referable to benign disease of the stump. This is, of course, an uncorrected figure and probably does not represent the true incidence because no attempt was made to follow the patients postoperatively. During 1939 at the Mayo Clinic, excision of the cervical stump was performed on twenty-eight patients.[3] From

1931 to 1941, we have operated upon forty-four women for abnormal conditions of the cervical stump. During this same ten-year period we performed 293 subtotal hysterectomies. These statistics suggest that this condition is not uncommon and it is probable that often enough the complaints of these patients have not been recognized as originating in the cervical stump.

SYMPTOMATOLOGY

Although relief from complaints arising in the uterus results *pari passu* from subtotal hysterectomy, those complaints from pre-existing cervicitis remain unchanged. Perhaps many surgeons fail to correlate vague pelvic complaints with cervicitis, and perform an elective subtotal hysterectomy. Removal of the body of the uterus does not improve the condition of the cervix, but, on the contrary, tends to induce continuation and extension of the infectious process.

Chronic cervicitis of the cervical stump presents the same clinical manifestations as this condition does before the uterus has been removed. The most constant symptoms are a persistent leukorrheal discharge, backache, pelvic pain and urinary discomfort. (Table I.) All forty-four patients in

TABLE I
SYMPTOMS

	No. of Cases
Leukorrheal discharge	44
Bloody discharge	14
Pelvic pain	20
Dyspareunia	9
Backache	21
Dysuria	20

our series were troubled with an abnormal vaginal discharge of varying amounts. Many women have been led to believe that

general infection disappeared following removal of the stump. One of these women gave a history of subtotal hysterectomy for

FIG. 1.   FIG. 2.

FIG. 1. Preoperatively the cervix was hypertrophied and chronically diseased with stenosis of the external os. The smallest sound could not be passed. This cervix was apparently normal at the time of subtotal hysterectomy eight years previously.
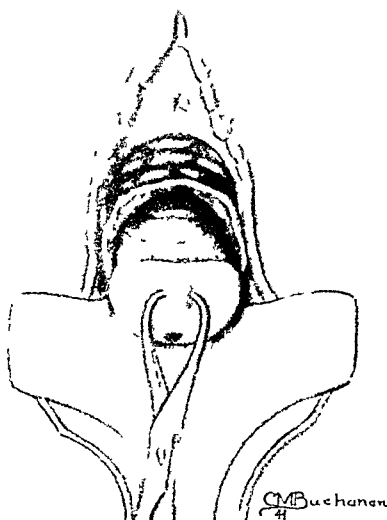
FIG. 2. A circular incision is made around the cervix and the mucous membrane is elevated, exposing Mackenrodt's ligaments. Dissection is continued upward separating first the bladder then the peritoneum from the anterior and superior aspects of the stump.

a vaginal discharge is not abnormal unless it contains blood. This is especially true in women in whom the menstrual function has been destroyed by hysterectomy. Whereas most of these patients presented mucous and watery discharges, fourteen reported spotting or frank vaginal bleeding. Needless to say, the latter were considerably alarmed.

Combinations of pelvic pain, backache and dyspareunia are frequent, resulting from lymphatic extension of the infectious process into ligaments supporting the cervix. Such pain can readily be reproduced on manipulation of the cervix during bimanual examinations. Dysuria and even marked prolapse of the stump are also encountered.

In certain cases the cervix, though sometimes least suspected, may be a focus of infection. In two of our cases all evidence of

fibroids eight years before. She came in complaining of severe abdominal pain. She had been treated unsuccessfully by several physicians, none of whom suspected the cervical stump. Physical examination revealed closure of the external os, hypertrophy of the cervix and retention of infected cervical secretions. The abdominal pain completely disappeared following removal of the cervical stump, and the patient's general health greatly improved. The other patient gave a similar history with a persistent low grade fever which was likewise eliminated by removing the stump. The improvement in the general well being of these patients after operation was remarkable.

MANAGEMENT

The general management of chronic cervicitis of the cervical stump does not

differ from the treatment of the infected cervix when the uterus has not been removed. General conservative therapy should be tried before radical procedures are contemplated. Conservative measures include dilatation and office cauterization. Such cauterization should be done only when erosion or eversion exists without extensive cervical hypertrophy and cystic changes. Some gynecologists have advocated deep cauterization[1] at the time of hysterectomy. This might impede the development of malignancy, but symptoms of chronic cervical infection will persist in many cases. Extensive cauterization is also undesirable because it predisposes to the formation of stenosis of the cervix, which interferes with drainage and causes retention of cervical secretions. Stenosis may follow cauterization of the stump following hysterectomy. In one of the cases reported this led to abscess formation. When nonoperative means prove unsuccessful, or in the presence of hypertrophy, stenosis or bleeding, surgical removal of the cervix is indicated. A Shroeder or Sturmdorf amputation or conization is not sufficient; the entire cervical stump should be extirpated. Removal of the cervical stump is a technically easy operation to perform. When the stump is removed, plastic procedures such as repair of cystoceles or rectoceles can and should be done.

TABLE II

Type of Operation	No. of Cases
Removal of stump	39
Fothergill operation (normal stump)	2
Radium application to stump (biopsy showed cancer)	1
Vaginectomy with removal of stump	2
Associated Procedures	
Hemorrhoidectomy	2
Anterior colporrhaphy	10
Posterior colporrhaphy	10
Cauterization of Skene's glands	1

TECHNIC

An elliptical incision is made around the cervix. The mucous membrane is then dissected free from Mackenrodt's ligaments laterally, from the pubocervical fascia anteriorly and from the peritoneum and ligamentous attachments superiorly. The peritoneum can usually be elevated without opening it. This obviates the danger of
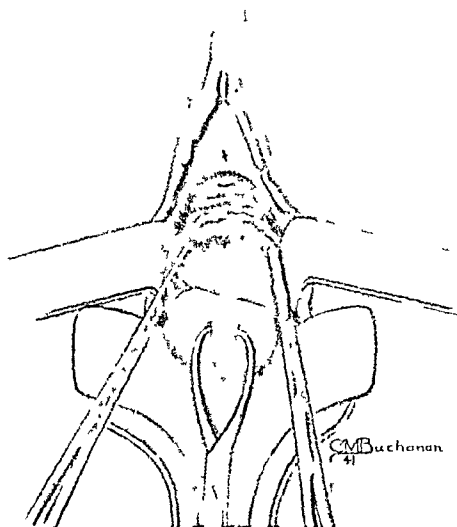

Fig. 3. Ochsner clamps are applied to Mackenrodt's ligaments, which are severed and ligated with transfixion ligatures. This frees the cervix almost completely.

injury to the intestines should they be adherent to the cervical stump. If necessary, the cul-de-sac can be opened from behind and the dissection continued anteriorly to prevent such injury. If this is done, suturing the uterosacral ligaments together will aid in supporting the vaginal vault and will prevent the formation of an enterocele. The wound is closed by suturing Mackenrodt's ligaments together and repairing the defect in the mucous membrane.

In the presence of prolapse of the cervical stump accompanied by cystocele and rectocele, the prolapse can be corrected by parametrial fixation together with the usual repair of the associated conditions. However, this should be done only if there is no other abnormal cervical condition than the prolapse.

COMMENT

An intensive analysis of the forty-four cases in this series cannot be made because it is believed that the series is too small to

form any generalities. However, a few statistics are of sufficient significance to prove informative. The average interval

ried women is unwarranted. The average age in our series was 44.2 years. It can readily be seen that these patients fall in
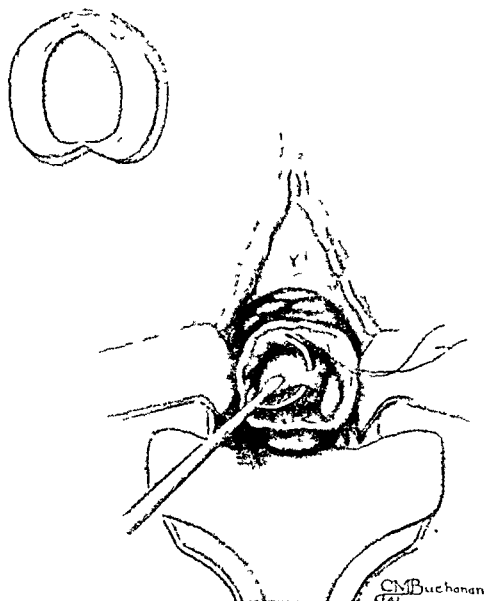

FIG. 4.   FIG. 5.

FIG. 4. The remaining ligamentous attachments of the stump are severed, and reconstruction of the vaginal vault is begun. Mackenrodt's ligaments are sutured together, to the pubocervical fascia and to the uterosacral ligaments. Reconstruction of these ligaments are necessary to prevent future prolapse of the vaginal vault. *Inset:* The extirpated cervical stump has been bisected, showing a dilated endocervical canal. This cavity was filled with mucopurulent secretion.

FIG. 5. To complete the operation, the defect in the mucous membrane is closed with a continuous suture. Drainage is not necessary.

between hysterectomy and removal of the cervical stump was 7.1 years. The exact significance of this figure is not known. This figure would suggest that the assumption that removal of the uterus will lead to atrophic changes in the cervix within a few years is erroneous. It would appear that in these cases disease of the cervix has been progressing during this interval to an extent requiring surgical excision. In many of these cases the patients have complained of symptoms for many years. The average parity was 2.9. The average interval between the birth of the last child and removal of the cervix was 12.5 years. It is interesting to note that three of the women in the series were not married. This suggests that possibly these unmarried women had had no vaginal examination prior to laparotomy, or that the assumption that chronic cervicitis does not exist in unmar-

the age group in which carcinoma is most likely to occur. In one case carcinoma was diagnosed by biopsy and radium was inserted into the cervical canal. In two of the remaining cases early malignant changes were found on microscopic examination. Removal of the cervix at this early stage was a valuable prophylactic measure for these two patients.

In one case subtotal hysterectomy was performed for uterine fibroids within two years of intra-uterine radium therapy. The cervix, removed a year and a half later, revealed active endometrial transplants.

A proper examination of the vagina and cervix should be conducted when a hysterectomy is contemplated and the decision as to total or subtotal operation should be made accordingly. If the surgeon believes that a total operation, although indicated, is too formidable to be done in certain

cases, the subtotal operation should be performed. The patient should then be reminded that the cervix has not been removed and that she should return for the removal of the diseased stump within a year. The increase in total hysterectomies over subtotal hysterectomies in the large clinics indicates that the undesirable effects of a retained cervical stump are becoming generally recognized.

There is no additional danger in removing the cervical stump than in doing any plastic operation on the vagina. There were no deaths in our series and the hospital course was short and uncomplicated in all cases.

SUMMARY

1. The importance of recognizing pathologic conditions of the cervical stump is emphasized.

2. The performance of complete hysterectomy in all indicated cases is urged.

3. The treatment of chronic diseases of the cervical stump is discussed and the technic of removal of the stump is described.

4. Forty-four cases of chronic cervicitis of the stump are briefly analyzed.

REFERENCES

1. CASHMAN, B. Z. The role of deep cauterization in the prevention of cancer of the cervix. A report of ten thousand cases. *Am. J. Obst. & Gynec.*, 41: 216, 1941.
2. CASHMAN, B. Z. and FRANK, J. S. Deep cauterization of the cervix—a factor in reducing the mortality of hysterectomy. *Surgery*, 9: 134, 1941.
3. COUNSELLER, V. S., MASSON, J. C. and WAUGH, J. M. Surgical report on gynecology for 1939. *Proc. Staff Meet., Mayo Clin.*, 15: 792, 1940.
4. COUNSELLER, V. S. The position of total hysterectomy in the treatment of benign and malignant conditions of the uterus. *Am. J. Obst. & Gynec.*, 37: 217, 1939.
5. HENRIKSEN, E. The retained cervical stump with special reference to nonmalignant changes. *Am. J. Obst. & Gynec.*, 37: 452, 1937.
6. MASSON, J. C. Total versus supravaginal hysterectomy. *Am. J. Surg.*, 48: 255, 1940.
7. McDONALD, E. P. Panhysterectomy. A study based on 2,773 consecutive hysterectomies. *New York State J. Med.*, 39: 503, 1939.

iiii. INFLUENCE OF ESTROGENS ON GENUINE PRE-ECLAMPSIA AND ECLAMPSIA

EVAN SHUTE, M.D.

LONDON, ONTARIO

THAT estrogens are helpful in the prophylaxis of true convulsive eclampsia has been claimed by both Siegler[1] and the author.[2][3] The basis for such treatment, of course, is the finding that in eclampsia the estrogen values in both blood and urine are low,[4,5] and the interesting experiments of Margaret Barrie[3] who produced eclampsia-like conditions in rats. The Smiths[6] have recently stated their belief that the low estogen level is the fundamental endocrine imbalance in eclampsia. It is probably unfortunate for the future of the hormone management of eclamptic toxemia that certain workers have, on this basis, begun to administer simultaneously prodigious doses of estrogens and progesterone, which are antagonistic substances.

To the previously published histories, the following case reports of eclamptics may be added. They substantiate what has been claimed for oestrogen therapy in this disaster of pregnancy:

CASE REPORTS

CASE I. (Courtesy Dr. W. Hughes.) This primipara, aged sixteen years, was first seen at 10 P.M. December 7, 1941. She had seemed normal until December 6, when she had had a one plus albuminuria. Her pressure then was 135/70. She was at term and had gone into labor spontaneously at 3.00 A.M. on December 6 at a hospital twenty-five miles from here. The pains subsided, only to return at 2.30 A.M. on December 7. She mentioned some headache and dizziness during the three hours prior to delivery and a mild stupor soon developed. She was given ½ cc. of pituitrin fifteen minutes before delivery. She had nembutal but no anesthetic for the labor, indicating the depth of her stupor. She finally delivered spontaneously at 8 A.M. on that day. The placenta showed no infarction. The child was a 7 pound 3 ounce normal child.

Her first convulsion came on at 12.30 noon and there were eight altogether before I saw her at 10.00 P.M. At that time there was no edema. She was restless, disoriented, but admitted no headaches nor eye symptoms. She had been given ⅙ gr. of morphine sulfate for her last convulsion at 8.00 P.M. There was no liver tenderness. There was acetone on the breath. Her blood pressure was 135/65.

A retention catheter was inserted at once, and 100,000 International Units of progynon B was injected intramuscularly at the same time. Doses of 50,000 units were ordered for 3.00 A.M., 8 A.M. and 12.00 noon next day. On account of the acidosis she was given 500 cc. of 10 per cent glucose intravenously at once. The catheter urine showed a two plus albuminuria but no red cells or bile. The blood estrogen[7] was weakly negative. I did not see her after that evening. At 1.20 A.M. she was given gr. ¼ morphia as there had been a convulsion at 1.00 A.M. She had an eight-minute long convulsion at 2.25 A.M. During that night her output was 28 ounces, almost free of albumen. At noon her pressure was 136/70. The intravenous glucose had been repeated at 10.00 A.M. by her own physician.

On the morning of December 9 there were three more brief convulsions, involving only the arms or face. She had had no estrogens since noon of December 8. Accordingly, she was given 5 mg. of stilbestrol three times on the ninth and twice on the tenth day. She was quite clear mentally during all that day and there were no more convulsions.

On December 10 a puerperal psychosis began. This cleared up by December 17 and she was discharged from hospital on December 20. She has since displayed some schizophrenic tendencies.

CASE II. This woman had previously had a term stillbirth, due to a hard forceps, and a spontaneous abortion. Her third pregnancy began on November 11, 1940, when she was

thirty-seven years old. When she was seen first on January 7, 1941, her weight was 163 pounds, her blood pressure 148/94 and her blood estrogen was positive. She began to vomit a great deal. This was controlled quite well by repeated hypodermic injections of testosterone propionate.[8] On March 10 her weight was 168 pounds, blood pressure 136 90, and there was no edema. On March 25 her pressure was 146/94 and the dose of wheat germ oil (Kelly's) she had been taking was accordingly raised to three tablespoons per day. On May 6 her weight was 177, her pressure had jumped to 170/96 and there was a moderate degree of edema of the legs.

I was called to see her in convulsions at 7.30 A.M. on May 21. Her sister had noticed a smoky urine for two days before and there had been great edema of the face, legs, and hands for four days before. I gave her gr. $\frac{1}{2}$ of morphia and sent her to the hospital. Her tongue was greatly swollen and she was stuporous. A retention catheter was inserted, and two ounces of smoky urine were obtained, containing 40 Gm. of albumin per liter. She was given 50,000 International Units of progynon B although her blood estrogen was still positive. The progynon B was repeated every four hours, so that she had four doses by nightfall. By 9.30 A.M. of May 21, she was rational, had no headache, but had blurred vision. At 11.00 A.M. she was sleeping quietly with no improvement in output. By 12.30 noon the output had begun to increase, she could read, and her tongue was only half as swollen as at first. She could remember everything up until 7.10 A.M. of that day.

At 8.00 P.M. the volume of urine output was steadily increasing and the urine was much less bloody, but the albumin was still 4 plus and 24 Gm. per liter, and her blood pressure was slowly rising. She seemed well and rational and had less edema of the eyelids. By 10.30 P.M. she was becoming restless and confused and her pressure had not dropped. A bag induction with rupture of the membranes was carried out under cyclopropane anesthesia. The child was alive at that time. Her pressure soon fell to 160/100 thereafter and she dozed quietly most of the night.

On the morning of May 22 she had no pains, and there was a fair amount of bloody urine output. There was much less edema generally. She felt and looked well. All day her pressure varied between 190/124 and 180/110. Strong

uterine contractions began at 3.00 P.M. The bag came out at 9.15 P.M. By version and extraction a six months female child was delivered at 10.30 P.M. The placenta seemed normal but was slightly adherent. Her pressure was 170/116 just after delivery. Next day there was only a one plus albuminuria, 3 to 5 Gm. per liter.

She made an uninterrupted recovery. She left the hospital on May 31 with a 2 plus albuminuria (catheter) and some hypertension. On both August 15, and October 20 there was still a trace of albumin in the urine. Her blood pressure on June 13, 1941, was 156/102. She felt well then and still does.

CASE III.   This woman, aged thirty, had had a stillborn premature baby after eclampsia and two live children after her next two toxic pregnancies. She was admitted to the hospital in a semiconscious state on February 18, 1942, at 2.45 A.M. She was not due until April 5, had had the usual antenatal care in the country, and had had a blood pressure of 160/100 and a slight ankle edema when she had last reported three days before. There had been great edema of the eyelids, blurred vision, uterine pain and "indigestion" the day before admission. A mustard plaster had been used for this. She had also bled from the vagina on that day. She later mentioned that there had been no fetal movement since the afternoon of that day. There had been a convulsion at midnight. At that time her physician in the country had administered $\frac{3}{8}$ gr. of morphine.

When she was seen by us she was wildly incoherent and threshing about. Her pressure was 90/70. There was a slight ankle edema. It took three people to hold her in bed and it was quite impossible to listen to the fetal heart. We stayed with her for two and one-half hours, and finally she became more manageable with an additional $\frac{3}{4}$ gr. of morphine. A retention catheter was inserted at the first, of course, and 100,000 International Units of progynon B were given intramuscularly. Her first 2 ounces of urine showed 4 plus albumin and 15 to 20 red cells and 30 to 50 white cells per low power field.

By 8.00 A.M. she was still somewhat frantic, and unfortunately got another $\frac{1}{4}$ gr. of morphine from the house officers. Her urine then showed only a 2 plus albumin. Her output had increased greatly but could not be measured as she had spilled into the bed after tearing out her catheter. At 9.00 A.M. she seemed nearly rational. By 10.00 A.M. she could count fingers and

henceforth was well oriented. Her blood pressure then was 106/90. She was given 5 mg. of stilbestrol every three hours that day, and daily thereafter, with no further sedatives. Opthalmoscopic examination of the fundi showed no abnormality. There was a slight edema of the abdominal wall. At 10.30 A.M. weak pains began but soon stopped. She slept much of the day. Her urine at noon had only a 2 plus albumin.

At 2.00 P.M. her pressure was 108/86 and 12 more ounces of urine had been obtained. There was much pus but almost no red cells. The fetal heart beat could not be heard. By 7.00 P.M. her output had been 8 ounces, her pressure was 120/86, and the membranes were ruptured artificially. Her speech was slightly thick and her tongue numb. There was old blood in the amniotic fluid. Weak pains soon began. At 11.00 P.M. her output was another 18 ounces and by 4.00 A.M. on February 19 another 38 ounces had been passed, with a 3 plus albumin, much pus, and some red cells.

She precipitated, at 5.35 A.M. of the same day, a stillborn female child, not macerated. The placenta was normal in appearance. At 10.00 A.M. her pressure was 135/90 and she seemed normal in every way. On the following day her blood pressure was 160/108 and on February 23 134/76. Her recovery was uninterrupted.

An intravenous estrogen of high potency is a real desideratum for these cases. This has been pointed out before[9] and certainly merits the consideration of the pharmaceutical houses. Estradiol benzoate given intramuscularly takes about seven hours to act, and much can happen to a convulsive patient in that time.

TRUE PRE-ECLAMPSIA

CASE IV. This patient, a primipara aged twenty-seven years, was first seen on May 22, 1940. Her last menses had begun on February 23. Her weight was 135, her blood pressure 106/76 and her urine was free of albumin. Her blood estrogen was positive and, therefore, she was put on a dram of Kelly's wheat germ oil daily. This was raised to a tablespoonful per day in September because of uterine tenderness and vaginal "pressure," and she went to Muskoka on a vacation. Suddenly, on September 20, she developed considerable edema extending up to the knees, a morning headache,

and some vague visual symptoms. There was no liver tenderness nor elevation of blood pressure. Her weight was 138 pounds. Her urine was clear. There was nothing to suggest either a nephritis or nephrosis.

Suspecting that the wheat germ oil was precipitating an early eclampsia,[3,10] we gave no more. After three and a half days, by which time all vitamin E effects should have worn off, the estrogen test was still negative; therefore, we concluded that she might do better on an estrogen. She was given 2 mg. of stilbestrol per day. Her symptoms and edema disappeared promptly. Late in October some leg edema recurred. On November 11, her weight was 155 and pressure 110/66, on 4 mg. of stilbestrol per day at that time.

On December 3, her blood pressure had risen to 140/100, there was a trace of albumin in the urine, her weight was 154, there was a slight leg edema, and she had begun to have morning headaches and spots before her eyes for the first time in months. Accordingly, her dose was raised to 5 mg. of stilbestrol per day. On December 5, there was no edema, no albuminuria, her pressure was 126/76, she felt well and she reported that she had had profuse urinations in the preceding two days. She had occasional spots before her eyes in the next nine days, but no headaches nor edema.

Labor began spontaneously on December 14. A maximally difficult breech delivery resulted in a fine 8 pound baby girl. Both mother and child did well.

On March 25, 1941, her weight was 146 and blood pressure 102/70. There was no albuminuria.

CASE V. This was an unmarried primipara, eighteen years of age, whose last menstrual date was April 28, 1941. She was first seen October 27, 1941, when her weight was 130 pounds, her pressure 110/64, her urine clear, and her blood estrogen negative. She had a completely uneventful pregnancy. On January 31, 1942, her weight was 135, her pressure 144/70 and her urine clear. There was no edema. On February 6, 1942, she began labor spontaneously at 10.00 A.M. She was sent to the hospital. By 5.00 P.M. she was dilated almost 7 cm., with moderate pains occurring every three to five minutes. At 8.30 P.M. she was 9 cm. dilated, and was drowsy between pains. She was completely oriented and had no headaches nor visual complaints. There were strong pains every two minutes but

no outcry. Indeed, she probably could have delivered without anesthesia. A catheter urine sample revealed but the faintest trace of albumin, but only two ounces was obtained, the first since admission. There were jerky little hand and arm movements if her body were touched and her reflexes were markedly hyperactive. Her blood pressure had jumped to 140/96. She was a direct O.D.P. at the outlet at 9.45 P.M. and accordingly a manual and forceps rotation and low forceps delivery were done. The child was an 8 pound 13 ounce boy and his shoulders were very difficult to extract. The placenta came in thirty minutes and showed only one, old white infarct 2 cm. in diameter near one margin, penetrating the placental substance.

She was given 100,000 International Units of progynon B during delivery and 50,000 units more at 1.00 A.M. Next morning she seemed normal in every way, had a blood pressure of 116/84, but a catheter urine showed a 1 plus albuminuria. She was put on 5 mg. of stilbestrol daily and made an easy convalesence.

CASE VI. This woman, aged forty years, had had two normal pregnancies, then a spontaneous abortion, and finally a macerated premature. Her last menstruation began November 12, 1939, and she was first seen February 2, 1940, when her weight was 142 pounds, urine clear, blood pressure 140/84 and blood estrogen negative. She was well until May 9 when her weight had risen to 155 and her pressure to 150/78; therefore, she was given 2 mg. of stilbestrol per day. On June 11, her weight was 156, her pressure 140/78, and she had a minimal leg edema, but a clear urine. Her stilbestrol dose was raised to 4 mg. on June 25. On July 2, her weight was 160, pressure 184/112 and her stilbestrol was reinforced with 50,000 International Units of progynon B. On July 5, her weight had fallen to 159, her pressure to 156/105 and her urine was still normal. She was put on a daily dose of 5 mg. of stilbestrol henceforth. She had 50,000 International Units of progynon B twice a week and by July 18 her weight was 162 and her pressure had dropped to 156/90. There was some leg edema at this time. On July 30, her weight was 164, her pressure was 176/104, there was considerable leg edema and the urine had a plus albumin for the first time. On August 15, her weight was 162, pressure 178/90 and there was a 2 plus albuminuria. She went into labor spontaneously on August

18, but was slow in getting under way; her membranes were artificially ruptured at 10.30 A.M. the following day. Strong pains began at 8.00 P.M. At noon on August 20 a 7 pound 10 ounce girl was delivered. The placenta came easily, displaying a white infarction of a cotyledon at one margin. She had some postpartum bleeding and severe shock, but thereafter made an easy convalescence.

On October 24, her pressure was 160/96. The following March she had an operation for glaucoma.

CASE VII. This primipara, aged twenty-eight years, menstruated last on July 20, 1940. When she was first seen on September 12 she had a blood pressure of 156/108, a clear urine, and weighed 132 pounds. Her blood estrogen was weakly negative. On October 21 her weight was 134, her pressure 138/90 and she was therefore given 2 mg. of stilbestrol per day. This dose was reduced to 1 mg. on October 28 when her blood pressure had fallen to 106/68, but was raised to 2 mg. again on January 27, when her weight was 155, pressure 114/76 and a minimal amount of leg edema had appeared. On March 13, the edema of the legs was very marked, her weight was 160, and her pressure 144/76. She was then taking 5 mg. per day of stilbestrol. On March 20, her weight was 163 and pressure 104/68. She went into labor spontaneously on March 27 with only moderate pains and was completely dilated in four hours. A boy weighing 6 pounds 9 ounces was soon delivered. The placenta came easily. There was one small, shallow, marginal crescent. There was some bleeding after I had left the hospital and therefore 1,000 cc. of intravenous glucose was given She made a good recovery. On May 26, 1941, her blood pressure was 140/86 and her weight 142 pounds.

CASE VIII. This woman had had one normal pregnancy. She was twenty-nine years old when seen on January 23, 1941, in her second pregnancy. Her last menstruation had begun on September 8. When seen she weighed 136 pounds, had a pressure of 126/80 and a negative blood estrogen, with clear urine. On February 20, her weight was 140, her pressure 150/88, her urine still was clear and there was no edema. She was put on 2 mg. of stilbestrol per day. On March 10, her weight was 145 and pressure 134/76, with no edema. On April 15, her weight was 150 and pressure 120/90, with slight leg edema. She was raised to 3 mg. of stilbestrol per

day. On May 22, her weight was 157, pressure 144/86, there was no edema and the urine was clear. She was raised to 5 mg. of stilbestrol per day. On June 12, her weight was 159, pressure 140/96, there was no edema, she felt well and the urine was clear. She was taken off salt and milk. On June 21, she precipitated, at home, an 8 pound 4 ounce normal female. The placenta was normal. Her convalescence was uneventful.

CASE IX. This patient was a primipara, aged twenty-one years. Her last menses began on March 2, 1940. When seen first on October 17 her weight was 166 pounds and her pressure 142/88; there was no edema, but a slight hydramnios, and the blood estrogen was a weak negative. Accordingly, she was put on 2 mg. of stilbestrol per day. This nauseated her. Accordingly, she was changed to 0.24 mg. theelol per day. On November 14, her weight was 172, pressure 134/82, and there was a trace of albuminuria. The hydramnios had gone. On November 21 she had a plus albuminuria, her pressure was 120/80 and there was slight edema of the ankles developing. Labor began spontaneously on December 11 at midnight. The pains were weak throughout. At 2.20 P.M. I did an extremely difficult midforceps and delivered a fine 9 pound 2 ounce boy. The placenta showed three very shallow whitish areas near the margin. There was slight postpartum hemorrhage. The next day her pressure was 144/86. She developed a mastitis after she left the hospital.

CASE X. This woman had had an induced abortion previously. When first seen on July 13, 1940, she was twenty-six years old. Her last menses had begun on March 23. Her weight was 118 pounds, her pressure was 114/60, her urine was clear, and her blood estrogen was negative. On October 1, her weight was 133 pounds, and her pressure had suddenly increased to 126/86. There was a slight edema of the fingers and legs. The blood estrin was again negative, and she was therefore given 2 mg. stilbestrol per day. On October 8, her pressure had dropped to 112/66 and she felt well. On November 9, her weight was 141, pressure 110/70 and she complained of hot flushes and spots before her eyes. On November 30, her weight was 144, her pressure 118/70, there was some edema of the fingers, and she admitted that she had been off the stilbestrol for a week. There were still spots before her eyes but no headaches; she said her head "pounded" when she was taking no stilbestrol. The blood estrogen again was nega-

tive. She was again given 2 mg. of stilbestrol per day.

On December 28, her weight was 151 pounds, her pressure 112/74, and there was slight edema of the legs and fingers. She had had great edema three days before, had raised her own dose to 8 mg. per day and volunteered the observation that a great increase in urine output had ensued. On January 7, her weight was 154 pounds, her pressure 114/60, she was "sleepy," had specks before her eyes, had slight edema and complained that her head felt "full"; and so was given 50,000 International Units of progynon B. She was induced on January 9 when ten days overdue with artificial rupture of the membranes. She had a low transverse arrest and forceps delivery of a boy weighing 7 pounds, 11 ounces. The placenta had one narrow, white, marginal crescent. She had an acute mastitis of both breasts after her discharge from the hospital.

CASE XI. (Courtesy of Col. W. Mace.) This patient, aged twenty-nine years, was a primipara. Her last menses began on November 15, 1940. When her physician joined the army she was sent to me. On July 10, 1941, her weight was 175 pounds, her blood pressure was 132/94, and there was a minimal edema of the legs and fingers. Her blood estrogen was negative; therefore, she was taken off salt and milk and given 2 mg. of stilboestrol per day on July 21. On July 29, her weight was 176, her pressure 134/94 and the edema was the same. There was some cholecystitis. Her urine was clear. The dose of stilbestrol was raised to 4 mg. per day. This was maintained until her delivery. On August 23, her weight was 180, her pressure 132/96, there was a trivial leg edema, and the urine was clear. Spontaneous labor began on August 24. Her pains were very sluggish and she was rested that night with morphia. In the morning her membranes were ruptured artificially. A forceps rotation was finally done and a 7 pound, 12 ounce boy was delivered. The placenta came easily. There was one tiny marginal crescent on the maternal surface. The recovery was uneventful.

Undoubtedly, in the light of later experience, many of these women should have had larger doses of estrogens.

DISCUSSION

It has been said "There are no hopeless situations; there are only men who have

grown hopeless about them." These case histories speak for themselves. A careful perusal of them will indicate the value of estrogens in the management of true pre-eclampsia and even in convulsive cases of eclampsia. It is to be noted that all the pre-eclamptics remained ambulant. This therapy should be, and is, more effective for pre-eclampsia than for eclampsia, for prophylaxis than for therapy. Therefore, the recognition of true pre-eclampsia is the crux of the situation. This is usually difficult on a purely clinical basis; a blood estrogen assay is a *sine qua non*. Those who object to the author's assay method[7] must find one that is more satisfactory and use it. Otherwise this therapeutic method will be discredited, for estrogens given to the vast majority of so-called pre-eclamptics can be of no assistance and may actually do harm, perhaps precipitating abruptio placentae.[2,11] Most so-called "pre-eclampsia" cases already possess too high an estrogen, should be given vitamin E, and run no risk whatever of terminating in convulsions. It is a pity that loose use of the term pre-eclampsia for every late toxemia of pregnancy should vitiate most of the literature on the subject.

SUMMARY

1. The case reports of three convulsive eclamptics and of eight true pre-eclamptics are presented in detail. One of the latter was induced and all remained ambulatory.

2. All were favorably influenced by the administration of estrogens.

3. This therapy is more effective in the prophylaxis than in the control of convulsive eclampsia, as could be anticipated.

4. A blood estrogen assay is a *sine qua non* in the recognition of genuine pre-eclampsia.

REFERENCES

1. SIEGLER, S. L. Estrogenic and chorionic gonadotrophic hormone in normal pregnancy and in toxaemia of pregnancy. *J. Lab. & Clin. Med.*, 24: 1277–1281, 1939.
2. SHUTE, E. V. The use of estrin in the treatment of eclampsia. *Endocrinology*, 21: 594–598, 1937.
3. Idem. The effect of estrogens on true pre-eclampsia and eclampsia. *Am J. Obst. & Gynec.*, 40: 1003–1011, 1940.
4. SMITH, G. V. S. and SMITH, O. W. Evidence for placental origin of excessive prolan of late pregnancy toxaemia and eclampsia. *Surg., Gynec. & Obst.*, 61: 175–183, 1935.
5. MUHLBOCK, O. Free and combined oestrogens in blood during pregnancy. *Lancet*, 1: 634, 1939.
6. SMITH, G. V. S. and SMITH, O. W. Estrogen and progestin metabolism in pregnancy; endocrine imbalance of pre-eclampsia and eclampsia. *J. Clin. Endocrinology*, 1: 470–476, 1941.
7. SHUTE, E. V. Determination of the estrogenic substance in blood serum by means of an estimation of the antiproteolytic power of the serum. *Am. J. Obst. & Gynec.*, 35: 970–977, 1938.
8. Idem. Hormone management of the nausea and vomiting of early pregnancy. *Am. J. Obst. & Gynec.*, 42: 490–492, 1941.
9. Idem. The diagnosis and treatment of the common disorders of menstruation. *Canad. M. A. J.*, 40: 38–43, 1939.
10. Idem. Vitamin E. Symposium. London, England, 1939.
11. Idem. The prophylaxis of abruptio placentae with vitamin E. *Surg., Gynec. and Obst.*, (in press).

ACUTE GASTRODUODENAL PERFORATION*

ROBERT F. BARBER, M.D.   AND   JOHN L. MADDEN, M.D.†

Director of Surgery, College Surgical Service, Kings   Resident in Surgery, College Surgical Service, Kings
County Hospital   County Hospital

BROOKLYN, NEW YORK

FROM June 1, 1937, to June 1, 1942, there were ninety-one patients with gastroduodenal perforations treated on the Long Island College Surgical Division of Kings County Hospital, Brooklyn. Eighty-six were treated by immediate operation and the remaining five conservatively. A consideration of the latter form of treatment with individual case reports is included.

During this period the majority of these operations were performed by the resident house staff and the cases presented are a consecutive group.

Incidence. During a five-year period there were 19,104 surgical admissions on this service, an annual average of 3,821. Within this same period ninety-one patients with perforation of a duodenal or gastric ulcer were treated, an average of fifteen per year. There was one perforated ulcer for every 210 admissions (0.47 per cent).

Seasonal Variation. The incidence of perforation was lowest during the autumn. Throughout the other seasons of the year there was a uniformity in the distribution. The variations are shown in Table 1.

Nationality. Nationalities were many and varied. The highest incidence of per-

foration was in the Irish, followed in close order by the Italian, Negro and Hebrew races, respectively.

Sex. Of ninety-one cases, only six were females, a ratio of fifteen to one (6.7 per cent).

TABLE II
NATIONALITIES

Nationality	No. of Cases
Irish	17
Italian	15
Negro	12
Hebrew	11
Norwegian	8
German	5
Swedish	4
English	3
Austrian	2
Greek	2
Canadian	1
Chinese	1
Polish	1
Scotch	1
Undetermined	7
Total	91

Age Group. The highest incidence of perforation was in the fourth decade. The fifth and sixth decades, respectively, were next highest. There were no perforations in the first two nor above the eighth decade. The youngest patient was a negro male of twenty-one and the oldest a man of seventy years. There were five cases of recurrent perforation. In three, both perforations occurred in the same decade, namely, one in the third decade and two in the fourth. In the remaining two the first perforation occurred in the fourth and fifth decades, the repeat perforations occurring in the fifth and sixth decades, respectively. There was an interval of five years between perforations in each of the last two cases. (Table III.)

TABLE I
SEASONAL VARIATIONS

Season	No. of Cases	Per Cent
Spring	26	28 5
Summer	25	27 5
Autumn	16	17 6
Winter	24	26 3

Age	No. of Cases	Deaths	Mortality, Per Cent
10–19	0	0	0 0
20–29	10	0	0 0
30–39	30	2	6 6
40–49	26	1	3 8
50–59	15	5	33 3
60–69	4	2	50 0
70–79	1	1	100 0
	86	11	12 8

Duration of Symptoms of Perforation. The duration of symptoms refers to the time interval between the immediate onset of symptoms and operation, and not to the interval between the onset of symptoms and admission to the hospital. There were seventy-six patients (86.4 per cent) operated upon within the first twelve hours of which forty-one (53.9 per cent) were within the first six hours, and thirty-five (46.1 per cent) in the second six hours. In one case symptoms extended over a period of fifty-nine hours. (Table IV.)

TABLE IV
DURATION OF SYMPTOMS IN OPERATIVE CASES

Duration of Symptoms— Hours	No. of Cases	Deaths	Mortality, Per Cent
0–6	35	0	0 0
6–12	39	7	18 0
12–18	9	3	33 3
18–24	1	0	0 0
24–36	1	1	100 0
36–48	0	0	0 0
48–60	1	0	0 0
	86	11	12 8

History of Previous Ulcer. There were fifteen cases in which no history of ulcer was obtainable. Three patients had vague complaints for one to two weeks prior to perforation, such as bloating and gaseous eructations. There were four others two of whom had symptoms of ulcer over a period of two weeks and two for three weeks. In five

patients a history of previous ulcer varying between a minimum of three to a maximum of seven months was obtained. In the remaining cases the history extended over periods varying between a minimum of one year and a maximum of twenty-five years. Two had symptoms of ulcer extending over a period of twenty-five years.

Previous Perforation. In each of five cases (5.1 per cent) there had been a previous repair of a perforated ulcer. Three of these operations were performed in this hospital and the interval of time between perforations was six, seven and twenty-nine months. In the remaining two the interval of time was five years in each case.

Predominant Symptoms. Pain, tenderness and muscle spasm were predominant symptoms. These occurred in every one of the cases with the exception of one in which there was pain and associated tenderness without accompanying muscle spasm. In the majority (86 per cent) the symptoms at onset were referable to the epigastrium or upper quadrants of the abdomen. In ten (11.6 per cent) the complaint on admission was pain in the right lower quadrant and in six of these the provisional diagnosis was acute appendicitis. Despite the findings of exquisite point tenderness and muscle spasm in the right lower quadrant, there was associated tenderness and spasm in the epigastrium or upper quadrants of the abdomen. This is of importance in differential diagnosis because in early cases of acute appendicitis with localization of symptoms in the right lower quadrant it is rare to have findings referable to the epigastrium or upper abdominal quadrants. In two cases the onset of symptoms was heralded by pain in the left lower quadrant, examination revealing exquisite tenderness and spasm in this region. The latter signs were also noted to a lesser degree in the left upper quadrant. Operation in each instance disclosed a perforation in the proximal portion of the body of the stomach with a free spill in the left lumbar gutter. These were the only cases in which perforation occurred in this region of the stomach

and on admission each presented identical symptoms and signs.

Shoulder pain was recorded in sixteen cases (18.6 per cent). Obliteration of liver dullness was noted thirty-seven times (43 per cent). Vomiting occurred in forty-one instances (47.6 per cent), was absent in twenty (23.2 per cent) and not recorded in twenty-five.

Shock is often spoken of as frequently accompanying gastroduodenal perforation. In this series, however, there were only four instances (4.4 per cent) in which the patient was in shock at the time of admission. There were no cases in which delayed shock supervened following hospitalization. In Table v the predominant symptoms are tabulated.

TABLE V

PREDOMINANT SYMPTOMS

	Present	Absent	No Record	Per Cent Present
I. Pain				
Abdominal	91	0	0	100 0
Shoulder radiation	22	6	63	24 2
II. Abdominal tenderness	91	0	0	100 0
III. Abdominal rigidity	90	1	0	98 9
IV. Liver dullness	24	43	24	26 4
V. Nausea	34	4	53	37 4
VI. Vomiting	44	22	25	48 5
VII. Shock	4	87	0	4 4

Location of Perforation. There were eighty-six instances in which perforation was recognized either at the time of operation or necropsy. Fifty-seven perforations were gastric, two of which involved the proximal portion of the body of the stomach. In twenty-four the perforation was duodenal, three involving the posterior wall. The smaller number of duodenal perforations is unusual, presenting a 2.4 to 1 ratio of gastric to duodenal. This is at variance with most series that have been reported. In three instances the location of the perforation was undetermined. In one it was stated that the perforation was an

equal distance on either side of the pyloric vein, and in another it involved the posterolateral wall of the jejunum immediately distal to the suspensory ligament (Treitz) of the duodenum. Three of the five patients with recurrent perforations had both operations performed on this service. In each instance the perforation was gastric.

TABLE VI

LOCATION OF PERFORATION

Location of Perforation	No. of Cases	Per Cent
Stomach	57	62 6
Duodenum	24	26 4
Jejunum	1	1 1
Multiple	0	0 0
Combined	1	1 1
Undetermined	3	3 3
Total .	86	

Size of Perforation. The uniformly small size of the perforations was characteristic as witnessed by the fact that in sixty cases (69.7 per cent) the diameter varied between 0 and 0.5 cm. In ten a variation between 0.5 and 1 cm. was present. The largest perforation was 3 cm. in diameter, a report of which may prove of interest:

This perforation occurred in a sixty-eight year old white male admitted to the hospital with an eight-hour history of symptoms referable to a gastric or duodenal ulcer. The perforation, associated with adjacent inflammatory edema and marked induration of the surrounding tissues, was located on the anterior wall of the pyloric portion of the stomach. Closure either by means of a purse string or through-and-through suture was impossible. A free graft of omentum was placed over the perforation, and the edges of the graft were sutured to the surrounding anterior wall of the stomach with five interrupted sutures of fine silk. A biopsy taken prior to repair of the perforation was reported as chronic inflammatory tissue.

The immediate postoperative course was uneventful, but he returned three months later complaining of persistent abdominal pain,

hematemesis, anorexia and an interval weight loss of twenty pounds. Gastric analysis revealed a high free acidity of 145 and a high total acidity of 165. On repeated gastric lavage there was no retention or evident bleeding. A gastrointestinal series revealed the presence of a duodenal ulcer. Upon review of the case a gastric resection was considered advisable. At the time of operation there was no evident ulceration and the only landmarks of the site of previous perforation were the five imbedded interrupted black silk sutures. There was a complete restoration of the whole thickness of the wall of the stomach without cicatrization. The rapidity of healing was remarkable. His postoperative course was uneventful. Since operation he has remained asymptomatic with marked improvement of appetite and a weight gain of thirty pounds. Table VII indicates the varying sizes in the perforations recorded.

In only five instances was the systolic blood pressure below 100.

In Table VIII the cases are listed according to the temperature, pulse and blood pressure at the time of admission.

Leukocyte Count. In seventy-three (84.8 per cent) of the eighty-six operative cases a leucocytosis was present. In eleven cases, the leucocyte count was within normal range, and in two there was a leukopenia. An increase in the polymorphonuclear leukocytes occurred on seventy-four occasions, was normal in eleven and below normal in one. Table IX gives a group classification of the degree of leukocytosis.

X-ray. Roentgenograms were taken for evidence of free air beneath the diaphragm in forty-one cases and were positive thirty-five times (85.3 per cent). Six cases were

TABLE VII
SIZE OF PERFORATION

Size of perforation	0–0 5 cm.	0 5–1 cm.	1–1 5 cm.	1 5–2 cm.	2–2 5 cm.	2 5–3 cm.	3–3 5 cm.
No. of cases	60	10	1	1	0	0	1
No measurements recorded	13						

Temperature, Pulse and Blood Pressure. There were fourteen instances in which the temperature was recorded as below normal, the lowest 96°F. In thirty-four cases the temperature varied between 99° and 100°F. In only eleven cases was the temperature over 101°F. In twenty-four of the patients the pulse varied between 70 and 100, exceeding 100 thirty-four times. In the majority of the cases, fifty-one (56.1 per cent), the systolic blood pressure varied between 110 mm. and 140 mm. of mercury.

negative, although at operation perforation was proved to be present.

Treatment. A total of ninety-one patients with gastric and duodenal perforation were treated on the service over a period of five years. In eighty-six cases the treatment was operative and in five conservative management was employed. In the operative group the preferred method of closure was by means of a purse-string suture of fine chromic catgut, re-enforced by an omental graft held in

TABLE VIII
VARIATIONS IN TEMPERATURE, PULSE AND BLOOD PRESSURE

Temperature	96–97°F.	97–98°F.	98–98.6°F.	98.6–99°F.	99–100°F.	100–101°F.	101–102°F.	102°+F.
No. of cases	1	4	7	8	34	25	11	1
Pulse	60–70	70–80	80–90	90–100	100–110	110–120	120+	
No. of cases	2	7	23	24	20	10	5	
Systolic blood pressure	70–80	80–90	90–100	100–110	110–120	120–130	130–140	140–150	150+
No. of cases	1	1	4	3	15	17	19	9	10

Blood pressure—not recorded—twelve cases

situ by interrupted sutures of fine silk. On occasion, silk interrupted sutures were used to close the perforation and an omental re-enforcement as above.

alternating with 5 per cent glucose in distilled water. This is continued until the fourth day, at which time a sufficient amount of fluid is taken by mouth to

TABLE IX
VARIATION IN LEUCOCYTE COUNT

White blood count	0–5,000	5–10,000	10–15,000	15–20,000	20–25,000	25,000+
No. of cases	2	11	26	28	17	2
Polymorphonuclear leucocytes	0–25%	25–50%	50–75%	75–100%		
No. of cases .	0	1	11	74		

Silk technic was used in the repair of the abdominal wound. Closure in layers without drainage was performed routinely with the exception of the occasional instance when the wound was closed with through-and-through sutures of heavy black silk.

Prior to operation a Levin tube is passed through either nostril into the stomach and fastened securely to the side of the face. The blood of the patient is typed and cross matched. Blood is always ready in our blood bank for immediate use. In the left antecubital vein an infusion of 5 per cent glucose in normal saline is started, and the patient is transferred to the operating room.

Upon return to the ward immediate gastric suction siphonage according to the method of Wangensteen is employed. Postoperatively, the suction is continuous for the first six hours. Feedings by mouth then follow according to the schedule in Table x.

The suction is alternately on and off every two hours during the next eighteen to twenty-four hours. The feedings are given at the start of the periods in which the suction is temporarily off. Subsequently, the time intervals are gradually varied so that by the evening of the second day or morning of the third day suction may be entirely discontinued and the stomach tube removed.

During the postoperative period the water and electrolyte balance is maintained by the daily parenteral administration of fluids, 5 per cent glucose in normal saline,

maintain an adequate water and chemical equilibrium.

Following operation for gastroduodenal perforation postoperative gastric dilatation is not an infrequent complication. It is easily recognized if kept in mind. It occurs on the average of forty-eight to sixty hours following the removal of the Levin tube and the characteristic symptoms which we have noted are nausea, anorexia, rapid pulse, restlessness, apprehension and free perspiration. The response to decompression of the stomach with a gastric tube is rapid. During the past year it has been the routine practice to perform nightly gastric lavage in all cases following the removal of the stomach tube. Lavage is repeated each night until a negative or minimal gastric residue is noted. This usually occurs between the fifth and seventh days postoperatively with some variations. By this method of postoperative treatment gastric dilatation as a serious complication has been eliminated.

CONSERVATIVE MANAGEMENT OF GASTRODUODENAL PERFORATION*

During the last year and one-half we have used elective conservative therapy in five cases, with satisfactory results. It merits further discussion. The questions naturally arise: What cases are to be treated in

* Since the preparation of this paper another case has been treated conservatively. The onset of symptoms was acute and typical. Physical findings presented a textbook picture of perforation. A complicating residual pelvic abscess was drained per rectum seven days after onset. Lesion of the duodenum was confirmed by roentgenogram.

TABLE X
ULCER DIET*
Feedings Instituted Six Hours Postoperatively

Day of operation:
Oz. 1 water alternating oz. 1 gelatin q2h

1st postoperative day:
Oz. 2 water alternating with oz. 2 gelatin q2h

2nd postoperative day:
Oz. 2 water
 " milk and cream ⎫ alternating q2h
 " gelatin ⎭

3rd postoperative day:
Oz. 4 water
 " milk and cream ⎫ alternating q2h
 " gelatin ⎭

4th postoperative day:
Oz. 5 water
 " milk and cream ⎫
 " gelatin ⎬ alternating q2h
 " gruel ⎭

5th postoperative day:
Oz. 6 water
 " gelatin
 " milk and cream ⎬ q2h
 " gruel
 " cream soup

6th postoperative day
Oz. 7 gruel
 gelatin
 cream ⎫ alternating q2h-sips of water
 cream soup ⎬ between feedings
 custard
 poached egg

7th postoperative day:
Oz. 8 milk and cream
 cereal
 egg
 cream soup ⎬ q2h
 puree vegetable
 custard
 poached egg
 mashed potato

8th postoperative day:
 Soft modified ulcer diet

9th postoperative day:
 Modified ulcer diet

10th postoperative day:
 Full ulcer diet

* The same dietary regime is used following subtotal gastric resection and has proven eminently satisfactory.

this manner? What are the indications for and against this mode of therapy? Unfortunately, the answers to these questions cannot be given in any didactic or clear-cut form so as to govern all cases. On the contrary, each case must be rigidly individualized. In general, it may be stated that the following points are taken into consideration before we judge a case suitable to conservative treatment: (1) Duration of symptoms; (2) general condition of the patient; (3) subjective symptoms and (4) objective findings on abdominal examination.

If the duration of symptoms is in excess of twelve hours and associated with subjective improvement (the patient will often state he feels better, that the pain is not as severe, etc.), nonoperative management is contemplated. Furthermore, if he appears to be resting comfortably and the findings on abdominal examination are not marked, conservative therapy is employed. This is in contrast to the individual who is admitted complaining of severe abdominal pain, progressive in character and associated with exquisite and diffuse abdominal tenderness and rigidity. In the latter instance immediate operation is mandatory. This type of patient often assumes a sitting posture to obtain relief and appears acutely ill. On the other hand, in the cases which were treated conservatively, the patient was resting comfortably in the supine position and did not appear so acutely ill.

That spontaneous closure of a perforation may occur is unquestionable. It has been the lot of almost every surgeon at operation to encounter instances of gastroduodenal perforation in which the protective barriers of nature had sealed the area of perforation. It was this finding which we have noted in Case 1. This led us to the belief that certain cases might well be selected for conservative management. It cannot be too strongly emphasized, that if such a condition presents itself at operation, the abdomen should be immediately closed and no attempt made to break down these protective barriers in order to disclose the site of perforation. At the expense of the well-being of the patient we have seen this performed with the resulting conversion of a localized inflammatory process into a diffusing peritonitis.

During the past year twenty cases were admitted with a perforation of a gastric or duodenal ulcer. In four (20 per cent) elective conservative management was

employed with favorable results. There were no deaths and follow-up study shows them to be well. The number of patients we have treated conservatively is small, the follow-up period is short. No final conclusion should be drawn. We simply desire to state we have treated five patients conservatively and each one recovered.

We remain of the opinion that all perforated ulcers are not of necessity to be treated by operation, but that selected cases may be treated conservatively with satisfactory results. Operative intervention, of course, remains the treatment of choice. If there is a doubt, immediate operation is performed.

Review of the cases in which the patients were treated without operation shows that on two occasions the duration of symptoms was less than twelve hours. Nevertheless, conservative management was employed. Interspersed between the cases treated conservatively there were others admitted with symptoms of the same duration or longer in which operation was performed because the clinical symptoms and signs which they presented on examination favored immediate operative intervention.

We believe that a review of the cases which were treated conservatively may be of interest:

CASE REPORTS

CASE I. In this case the treatment was both operative and conservative. The outcome aroused our interest in the possibility of selecting certain cases for nonoperative management.

H. J., a white male, age forty-three, was admitted on June 15, 1941, with a history of abdominal pain, nausea and associated vomiting of twelve hours' duration. Previous history relative to ulcer was vague. Physical examination revealed a well developed, well nourished white male lying quietly in bed, not appearing acutely or chronically ill. His temperature was 102°F., pulse 86, blood pressure 120/80. Positive findings were referable to the abdomen.

The abdomen was rounded and there were no scars. Tenderness and spasm were present in the right lower quadrant and to a lesser degree in the epigastrium. There was no evident obliteration of liver dullness, no masses, or palpable viscera. Rectal examination revealed tenderness high up on the right side. *Diagnosis:* Acute appendicitis.

The leucocyte count was 13,000, 88 per cent polymorphonuclear leucocytes. Roentgenographic examination revealed the presence of free air beneath the dome of the right side of the diaphragm.

At operation on the day of admission the peritoneal cavity was entered through an upper right rectus muscle splitting incision. Approximately 100 cc. of free cloudy straw colored peritoneal fluid was present. The stomach was found adherent to the inferior surface of the right lobe of the liver. At the point of junction of the liver edge with the anterior wall of the stomach and duodenum there was a thin, filmy exudate which sealed the opposed surfaces. Cultures were taken and the wound closed in layers without drainage.

Postoperative treatment consisted of continuous gastric siphonage for a period of forty-eight hours and parenteral administration of fluids. On the second postoperative day routine postoperative ulcer dietary régime was instituted, with supplementary parenteral fluid administration to maintain an adequate water balance. The postoperative course was uneventful and he was discharged on the fifteenth postoperative day.

Comment. The condition of the patient at the time of admission appeared satisfactory. He was resting comfortably, appeared to be in no acute distress and felt subjectively improved. However, due to the presence of tenderness and moderate spasm in the epigastrium, and the roentgenographic evidence of free air beneath the dome of the diaphragm on the right side, operation was advisable. At operation the area of perforation was so well walled off that any attempt to locate the perforation by breaking down the protective barriers already present was thought inadvisable.

CASE II. F. E., a thirty-four year old white male, was admitted on August 3, 1941, with a history of onset of epigastric pain twenty-two hours prior to admission. The pain became diffuse and rapidly increased in severity; it

was associated with nausea but no vomiting. Past history revealed symptoms referable to ulcer of ten years' duration, with increasing severity of symptoms during the two years before the present admission.

Physical examination revealed a well developed, well nourished while male appearing acutely ill. His temperature was 100.6°F., pulse 80. Positive findings at the time of examination were limited to the abdomen.

The abdomen was flat, muscular and bore no scars. There was tenderness throughout with associated rebound phenomenon and moderate muscle spasm. Liver dullness was obliterated. *Diagnosis:* Perforation of a gastric or duodenal ulcer.

The white blood cell count 13,200, 76 per cent polymorphonuclear leucocytes. A roentgenogram revealed free air beneath the dome of the diaphragm on the right side.

The treatment was conservative, consisting in sedation, parenteral administration of fluids, and continuous gastric siphonage for forty-eight hours. On the second day following admission a routine postoperative ulcer diet was instituted with supplementary parenteral administration of fluids. His course in the hospital was uneventful. Local abdominal findings present on admission had completely disappeared on the fourth day of hospitalization. Gastric analysis revealed the presence of a hyperacidity with a high free hydrochloric acid of 145 and a high total of 165. A gastrointestinal series prior to discharge showed a constant deformity in the first portion of the duodenum. *Diagnosis:* Duodenal ulcer. He was discharged August 14, 1941.

Comment. Because of the duration of symptoms prior to hospitalization and the fact that he was handling his condition satisfactorily at the time of admission, conservative therapy was considered the preferred method of treatment. Follow-up examination over a period of eight months shows that he is maintaining the prescribed diet and has remained symptom free.

CASE III. J. W., a white male, age fifty-eight, was admitted October 14, 1941, with a history of being awakened at 2 A.M. on that day with severe epigastric pain with radiation to the right shoulder. Two hours following onset of symptoms he took whiskey and soda

without relief and was admitted to the hospital at 6:00 A.M. on the same day.

Physical examination at this time revealed a well developed, well nourished white male lying flat in bed, with positive findings limited to the abdomen. His temperature was 98.6°F., pulse 96, blood pressure 130/80.

The abdomen was flat with exquisite upper abdominal tenderness and rigidity. Marked tenderness and spasm were present in the right lower quadrant. Liver dullness was diminished. *Diagnosis:* Perforation of a gastric or duodenal ulcer.

The white blood cell count was 18,600 with 85 per cent polymorphonuclear leucocytes. Roentgenographic examination revealed the presence of free air under the leaflet of the diaphragm on the right side.

Six hours following admission, which was twelve hours following the onset of illness mild tenderness persisted in the epigastric region without associated muscle rigidity. No evidence of shock was present and his general condition was good. On October 16, his temperature was 100°F. and the patient was resting comfortably; he had no complaints and his chest was clear. There was very slight tenderness noted in the lower quadrants of the abdomen; no spasm or rebound tenderness were present. On October 18, the abdomen was soft; there were no tenderness or spasm, and he was accepting feedings well. Improvement was progressive, and the patient was discharged on October 24 ambulatory and without complaints. The hospital stay was ten days. Subsequent gastrointestinal series revealed the presence of a duodenal ulcer.

Comment. Examination within one hour after admission showed marked improvement in his condition. Tenderness in the epigastrium and right upper quadrant with moderate muscle spasm persisted but was less severe. Definite subjective and objective improvement were present. A trial of conservative therapy was thought permissible and rapid and progressive improvement in his condition occurred. To date he remains symptom free on a prescribed ulcer diet.

CASE IV. G. M., a seventy-four year old white male with no previous ulcer history, was admitted on January 28, 1941, with a history

of epigastric pain of nine hours' duration. No vomiting or nausea were present; there was no shoulder pain.

Physical examination revealed a well developed, well nourished healthy white male appearing acutely ill, but resting comfortably. His temperature was 101.6°F., pulse 80, blood pressure 130/80.

The abdomen was rounded and bore no scars; there was tenderness in the upper part of the abdomen most marked in the epigastrium with associated mild muscle spasm of an apparent voluntary type. There was no obliteration of liver dullness.

The white blood cell count was 9,900. Roentgenographic examination revealed evidence of free air under the dome of the diaphragm on the right side. *Diagnosis:* (1) Questionable perforation of gastric or duodenal ulcer; (2) perforation of a neoplasm of colon.

Conservative management was followed by progressive improvement in his condition. Roentgenographic examination on the seventh day following admission revealed absorption of the previously described free air beneath the diaphragm. Barium enema was negative. Gastrointestinal series revealed evidence of a duodenal ulcer.

Comment. The diagnosis in this case on admission was undetermined. Following the demonstration of free air beneath the diaphragm by roentgenographic examination the possibility of a perforated ulcer or a perforated neoplasm of the colon was entertained, the latter due to the fact that a history of recent change in bowel habit was obtained. Due to the apparent good condition of the patient conservative therapy was advisable. He remains symptom free, one and one-half years following his discharge from the hospital.

CASE V. W. H., a negro male, age sixty-one, was admitted on March 2, 1942 with a six weeks history of sharp, intermittent abdominal pains occurring two to three times a day and lasting one to two minutes. At 1:30 A.M. on the day of admission, shortly following ingestion of two drinks of whiskey and beer he noted the sudden onset of severe epigastric pain with associated nausea and vomiting. The pain caused him to double up, radiated to the left

shoulder and was accentuated upon respiration. He was admitted to the hospital five hours following the onset of symptoms.

Physical examination revealed a well developed, well nourished, obese colored male appearing acutely ill. His temperature was 100.2°F., pulse 108, blood pressure 124/80; white blood cell count 16,000, 87 per cent polymorphonuclear leucocytes.

TABLE XI
WOUND HEALING

	No Drainage	Drainage	Total	Per Cent
No. of cases...........	76	10	86	
Wound healing				
Per primam..........	63	2	65	75.6
Per secundum........	6	4	10	11.6
Undetermined as result of death...........	7	4	11	12.8
Wound infection........	6	4	10	11.6

His abdomen was rounded but bore no scars. There were marked tenderness and spasm in the epigastrium and both upper quadrants. Tenderness also was present in the lower quadrants of the abdomen, but no spasm was elicited. Roentgenographic study revealed free air beneath the diaphragm on the right side. *Diagnosis:* Perforation of a gastric or duodenal ulcer.

Conservative treatment was employed. Six hours following admission the patient had no complaints. No change in abdominal findings was present. Within twelve hours marked subjective and objective improvement was noted. On March 5, moderate distention of the abdomen was present without associated tenderness or spasm. The distention became progressively more severe with difficulty in respiration. On March 9, there was clinical evidence of pneumonic consolidation in the left lower lobe. The same day a copious diarrhea occurred. The abdomen was soft, flat and nontender. On March 14, his condition was greatly improved. Rectal examination at this time revealed no evidence of a pelvic abscess. The temperature varied between 100.2° and 103.6°F. for the first eight days following his admission, gradually returning to normal on March 20 where it remained until the time of discharge on April 11, 1942.

The immediate treatment consisted of continuous gastric siphonage, sedation, and the parenteral administration of fluids. On March 3, feedings by mouth were started but were discontinued on March 6 due to the marked distention of the abdomen which was present. The subsequent treatment consisted in blood transfusion, oxygen administration (tent) and sulfadiazine therapy. A satisfactory response occurred.

The gastrointestinal series on April 3 revealed the presence of a duodenal ulcer. Gastric analysis on April 8 showed normal values.

Comments. The response in this case to conservative therapy was quite satisfactory until the third day following admission. Then moderate distention occurred at which time there was complete absence of abdominal tenderness and spasm. Distention was progressive over a period of four days with a complicating lobar pneumonia. Condition responded to prescribed treatment although the convalescence was stormy. On follow-up he remains asymptomatic and is maintaining prescribed ulcer diet.

CASE VI. D. R., a white male, age fifty-one, was admitted on August 29, 1941, with a history of sudden onset of severe generalized abdominal pain sixteen hours prior to admission. The pain caused him to double up, and was most marked in the epigastrium and umbilical region, and of an increasing severity. No previous history of ulcer was obtained.

Examination at the time of admission revealed a well developed, well nourished white male appearing acutely ill. His temperature was 101°F., pulse 64, blood pressure 94/50.

The abdomen was scaphoid with tenderness and boardlike rigidity in the epigastrium and upper quadrants. Tenderness without spasm was present in the left lower quadrant. *Diagnosis:* Perforation of a gastric or duodenal ulcer. Roentgenograms were not taken at the time of admission for evidence of free air beneath the diaphragm.

Routine conservative therapy was instituted followed by rapid and progressive improvement. Abdominal tenderness and rigidity had completely disappeared by the third day of hospitalization. Gastric analysis revealed a moderate hyperacidity and a gastrointestinal series demonstrated the presence of a duodenal ulcer. Hospital stay was twenty-four days.

Comments. Due to the long duration of symptoms prior to admission and with the general condition of the patient satisfactory, conservative therapy was the treatment of choice, with complete relief of symptoms and signs within three days following admission. This is the only case presented in which roentgenograms were not taken to demonstrate the presence of free air beneath the diaphragm.

Drainage. Insertion of drains into the peritoneal cavity or wound is not advocated. In the eighty-six cases in which operation was performed drainage was omitted in seventy-six of the patients, and used in the remaining ten. The cases in which drainage was not used, primary wound healing was obtained in sixty-three (82.9 per cent). In two cases there was no statement regarding the character of the wound healing. Seven of the patients who were not drained died, and thus wound healing was not determined. In the ten cases in which drainage was instituted wound healing was stated to be primary in two and secondary in four. Four of the patients who were drained died and again as above the character of the wound healing was not determined. Wound infections occurred in ten cases, a percentage of 11.6 per cent. The sulfonamide group of drugs was used locally in the peritoneal cavity and also in the wound prior to closure in only five instances and no conclusions are drawn.

Complications. There were no complications in fifty-four of the cases. In the remaining the complications were chiefly pulmonary in nature, with wound infections and peritonitis, respectively, the next in frequency. Several of the patients in this series had more than one complication. One of the patients developed in sequence a postoperative wound infection, pelvic abscess, lung abscess, bronchopleural fistula and empyema with recovery

after a hospital stay of eighty-nine days. In Table XII there will be found a list of the complications encountered following operation for perforation of gastric or duodenal ulcer.

TABLE XII
COMPLICATIONS

I Complications		
A Pulmonary		
Bronchopneumonia	6	
Lobar pneumonia	1	
Atelectasis	3	
Pulmonary edema	1	
Lung abscess	2	
Bronchopleural fistula	1	
Empyema	1	
Tuberculosis	1	
B Wound		
Infections	10	
Dehiscence	4	
C Peritonitis	8	
Pelvic abscess	1	
D Miscellaneous		
Gastrointestinal hemorrhage	3	
Pylephlebitis	1	
Parotitis, bilateral	1	
Delirium tremens	1	
Dextrocardia	1	
Syphilis	1	
E. Deaths	11	
II No complications	54 (62 8%)	

Hospital Stay. The shortest period of hospitalization following operation in the patients who survived was ten days and the longest period eighty-nine days. The average stay was 17.7 days. In the operative patients who succumbed (eleven) the shortest period of survival was one day, and the longest fifty days, with an average survival period of 8.5 days.

Mortality. There were eleven deaths in eighty-six operative cases, with a resultant mortality of 12.8 per cent. This is qualified by dividing the cases into two groups: first, according to age, and second, according to the duration of symptoms. Reviewing the former it is seen that the largest number of deaths numbering five out of fifteen cases (33 per cent) occurred in the sixth decade. This was almost half of the total number of deaths recorded, namely, eleven. Reviewing the latter it may be seen that seventy-four (86 per cent) of the patients were operated upon within twelve hours following the onset

of symptoms. There were thirty-five cases (40.7 per cent) in the first six hours without a death, and thirty-nine cases (45.3 per cent) in the second six hours with seven deaths or a mortality rate of 18 per cent. It is apparent from these statistics that, in general, the younger the patient and the shorter the time between perforation and operation, the better is the prognosis. The longest interval between the onset of symptoms and operation was fifty-nine hours, recovery ensuing. There were no deaths in the five patients who received conservative treatment. This number is too small to justify any conclusions.

Follow-up Study. We have been able to trace twenty-eight cases: twenty-seven males and one female, and this covers a postoperative period varying between two months and five years.

Results obtained may be classified as satisfactory and unsatisfactory. In one of the cases in which a satisfactory result was recorded it was noted at the time of his last follow-up examination, which was two and one-half years following repair of his perforation, that for the previous six months he partook of ten glasses (10 oz.) of beer and ten glasses (1 oz.) of whiskey between 12 o'clock midnight and 4:00 A.M. in the morning. His occupation was that of a defense worker in a machine-shop and he worked from 3:30 P.M. to 11:30 P.M., and slept on the average of four out of twenty-four hours. Despite this he remains symptom free. In May, 1942, a gastrointestinal series demonstrated the presence of a duodenal ulcer.

There were nineteen cases classified as satisfactory and nine as unsatisfactory. In the former group are included all of the patients who were treated conservatively. In the latter there were two cases which had repeat perforations within six and seven months, respectively. In each instance following the first operation periodic attacks of pain and nausea with associated vomiting occurred. Following the second repair one of the patients remained asymptomatic for four months, after which he

noted attacks of pain with vomiting every three or four weeks over a period of three months. These symptoms abated spontaneously, since which time he has been well. In the remaining case the patient still has recurrent attacks of abdominal pain, nausea and irregular attacks of vomiting. In four cases subsequent gastric resection was performed for persistence of symptoms. Three of the patients had resection performed within three months following the perforation, and the remaining patient one year following the perforation. Follow-up of these cases revealed the patients under dietary management to be completely asymptomatic. The remaining two of the unsatisfactory group have been followed for three years. One has never felt well since the operation, despite adequate and persistent medical treatment. The other felt well for one year, following which there was periodic recurrence of pain unrelieved by medical care. Both of these cases are incapacitated relative to their normal occupation.

SUMMARY

1. Ninety-one cases of gastroduodenal perforation have been reported, eighty-six treated by operation and five by elective conservative management.

2. Fifty-seven cases were gastric in origin and in twenty-four duodenal. In three the location was undetermined; in another it was in the jejunum and in one it was equidistant on either side of the pyloric vein.

3. The predominant symptoms and signs have been reviewed. The similarity in some cases to acute appendicitis and points of differentiation from the latter have been discussed.

4. A review of the cases referable to age, duration of symptoms, and mortality has been presented.

5. Shock was rarely associated with perforation. It was present in only four (4.4 per cent) of the cases reported.

6. Roentgenograms were taken for free air beneath the diaphragm in forty-one instances and was positive in thirty-five cases (85.3 per cent).

7. The order of frequency of postoperative complications were respectively, pulmonary, wound infections and peritonitis. In fifty-four of the cases there were no complications.

8. Drainage of the peritoneum or the wound is not recommended.

9. The immediate preoperative and postoperative treatment of the operative cases is considered.

10. A detailed discussion of the conservative management of gastroduodenal perforation has been given with individual case reports of the patients treated in this manner.

11. In eighty-six patients operated upon there were eleven deaths (12.8%). There were no deaths in the five patients treated conservatively. In general the variation in mortality is in direct proportion to the age of the patient and the duration of symptoms.

12. A follow-up study in twenty-eight of the operative cases is presented.

This includes both operative and nonoperative cases.

SNAPPING HIP

FIRST LIEUT. HOWARD DUDGEON, JR., M.C.

WACO, TEXAS

THERE is not much mention of the snapping hip in the general surgical textbooks. In fact, most of them pass this condition over with only three to four lines which fail to enlighten one as to exactly what it is or how to treat it. The literature is not very large and most articles report only one or two cases seen. I believe that there must be many of these hips seen, especially in industrial practice and among young active males, but these are either being overlooked or not diagnosed. The symptomatic cases manifested by pain and limp can be corrected by a simple, safe operation which gives almost 100 per cent cures.

I have six cases to report, two of which were seen by me in association with Dr. Fred A. Turner. The other four were seen on the Orthopedic Service of the Station Hospital, Camp Bowie, Texas. Out of 2,600 cases seen on this service since January 1, 1941, these four had all of the definite symptoms of snapping hip, an incidence of 1 per 650 admissions. All six cases seen were young males.

History. The history of this condition is very indefinite so far as the early medical literature is concerned; so I shall pass over it without further discussion.

Etiology. There is some question as to the etiology of this condition. Some claim it may possibly be due to an inflamed bursa about the hip joint or trochanter, malformation of the hip joint, tic or spasm of muscles, either voluntary or involuntary, or muscular relaxation, especially of the gluteus maximus. While Mayer, Pruitt, Lovett and DaCosta believe it is due to iliotibial tract abnormality.

I consider the cause to be due to trauma to either the iliotibial tract or the tensor fasciae femoris and possibly at times to the insertion of the gluteus medius muscle. However, the majority of the cases follow injury to the iliotibial tract alone.

Cases I, II, III and IV have definite histories of hip injuries which were later followed by the appearance of snapping in the injured hip. Case VI gives an indefinite history of injury, while Case V has had no injury so far as I could find out.

I believed that these men received vertical tears in the iliotibial tract in the region of the greater trochanter and that the healed scar in the tract formed a hard ridge which whips back and forth over the greater trochanter with each step. This eventually causes a bursitis over the trochanter which causes both local and referred pain.

Symptoms. The symptoms are very simple. The patient complains of a snapping, which he can feel and occasionally hear, in the region of the greater trochanter. There may or may not be pain. It was present in four of my cases. Pain is always present in the region of the greater trochanter and may be referred down the anterolateral aspect of the thigh to the knee. Two patients (Cases II and III) thought that the effected leg was shorter than the other. In fact, Case II had been told that he had a dislocated hip.

Physical Findings. There is a palpable "cord" whipping over the greater trochanter which goes anterior to on flexion of thigh and posterior to the trochanter on extension. This may be easily felt by placing the hand on the trochanter and having the patient walk. It is usually present with each step. Occasionally the "cord" may be seen to jump under the skin during locomotion. In old cases with severe pain and muscle spasm (Case II) there may be atrophy of thigh muscles and slight scoliosis as the patient favors the leg to ease pain.

Some patients lie in bed so that the injured leg seems upon observation to be shorter than the uninjured one. Measurement from the anterosuperior spine of the ilium to the internal malleolus will show that both legs are of equal length. An anteroposterior and oblique views of hip should be taken. In true snapping hip there is no bone change.

Diagnosis. A diagnosis is made by: (1) Palpable snapping of band over the greater trochanter upon flexion and extension of the hip; (2) pain in the region of the trochanter may or may not be present. There may be occasional referred pain to the anterolateral surface of the thigh; (3) no bone changes are shown by x-ray; (4) legs are of equal length; and (5) usually there is a definite history of trauma either remote or recent (four of my cases), but absence of this will not rule out the correct diagnosis.

Differential Diagnosis. This must be made from osteochondroma of the trochanteric region, intra-articular lesions such as loose bodies or osteochondromatosis, or subluxation associated with paralysis of the muscles of the hip. All, with the exception of muscle paralysis, show positive x-ray findings. Muscle paralysis may be differentiated by history, muscular weakness, and physical findings.

Treatment. The correction is surgical if pain and disability are present. If there is only snapping without pain and disability, no surgery is indicated as an explanation of the cause will usually satisfy the patient.

The operation should be done under local anesthetic as the tense band is found with difficulty if the muscles are fully relaxed by inhalation anesthesia. Dickinson reports a case of bilateral snapping hip which required five operations, three under ether, and the last two, which were successful, under local anesthesia. It is very important that the patient be conscious and able to snap the hip during operation, otherwise the band will often not be found and the patient will not be cured.

The technic used was that described by Campbell which is as follows, (Figs. 1, 2, 3 and 4): "An incision is made in line with the junction of the gluteus medius and tensor fasciae femoris muscles to the posterior border of the greater trochanter, thence continued distally in the longitudinal axis of the thigh a distance of 4 inches. On incising the ilio-tibial band, a definite, thickened strip may be palpated on its posterior inner surface. This portion is dissected up to the trochanter forming a flap 3–4" in length. The distal end of the tensor fascia femoris is then freed. The distal half of the fascial flap is cut off and the remaining fascial flap is transferred anterior to the greater trochanter, sutured to the fascia on the anteriolateral aspect of the thigh and fixed by chromic sutures." (Fig. 4.)

The leg is not immobilized in a cast as was formerly the practice, but a snug, tight dressing is applied to prevent any postoperative oozing. It is a good idea to use a soft rubber drain for a few days as there is a large area of skin that has been undermined. Exercise should be started as soon as the wound has healed.

Several other operations have been suggested by Binnie, Jones, Mayer and Pruitt, but they are essentially the same as the above described technic and the results also are the same.

CASE REPORTS

CASE I. E. A. D., age seventeen, a male, while playing football six months ago fell down pinning his left leg under him. There was sharp pain at the time of injury but not disabling. On the next day his hip and lateral side of thigh were sore but he soon recovered and after ten days was able to resume play. Three months after injury he began to have snapping of hip when he walked. At first there was no pain associated with this, but as snapping became more severe there was associated pain in the anterior and lateral side of thigh which extended down to the knee. The patient believed his left leg was shorter than the right and he favors it. He was very worried about the leg and thought that he may be crippled and unable to play football. Physical examina-

tion showed a typical snapping hip. Legs were of equal length; there were 1½ inches of

resected. The patient recovered completely and his football ability was not impaired.

FIG. 1.

incision

greater trochanter

FIG. 2.

greater trochanter

fascial scar
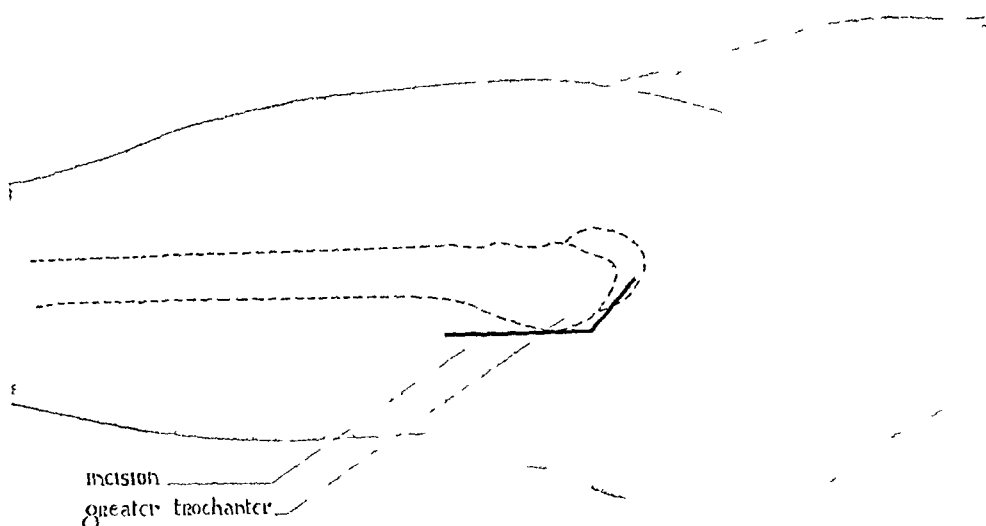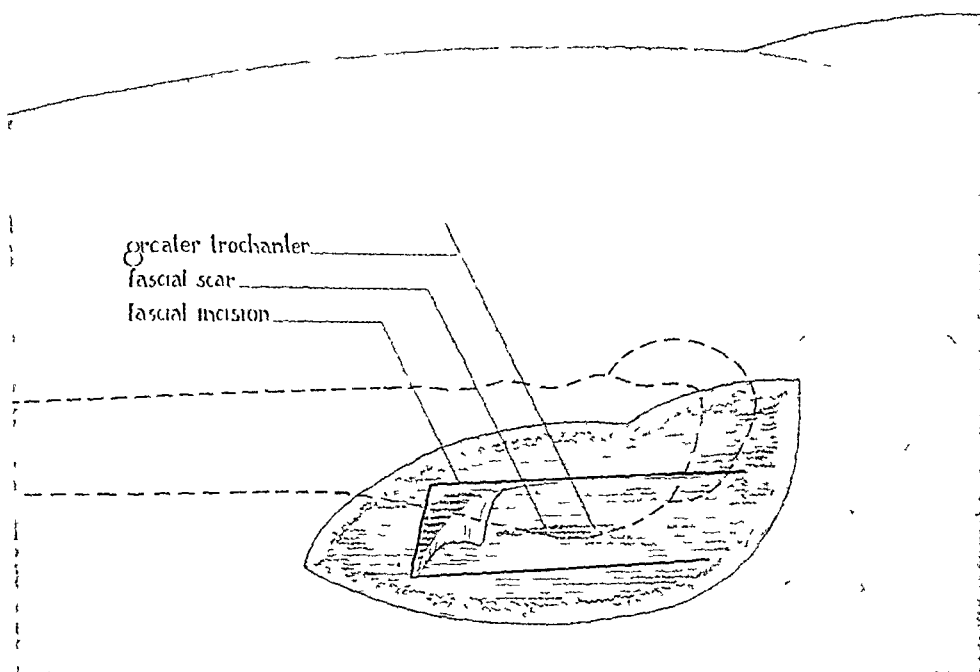
fascial incision

FIG. 1. Skin incision is shaped like a hockey stick and is made in line with the junction of the gluteus medius and tensor fascia femoris muscles to the border of the greater trochanter, then distally and parallel to the femur for about four inches.

FIG. 2. Two parallel incisions are made in the iliotibial tract and extended up to and above the greater trochanter where the gluteus medius and tensor fascia femoris gradually fuse into the tract. The thickened fascial band, which causes the snapping, is shown in the drawing and can be felt on the posterior inner surface of the iliotibial tract as a thickened cord.

atrophy in the left thigh; X-ray was negative. Operation was carried out under local anesthesia and the thickened iliotibial band

CASE II.   O. C. T., age thirty, injured his hip when he slipped and fell while running to the mess hall. His left leg was twisted. The regi-

fascia cut off here

greater trochanter

lateral scar

reflected fascial flap

FIG. 3.

sutured fascial stump
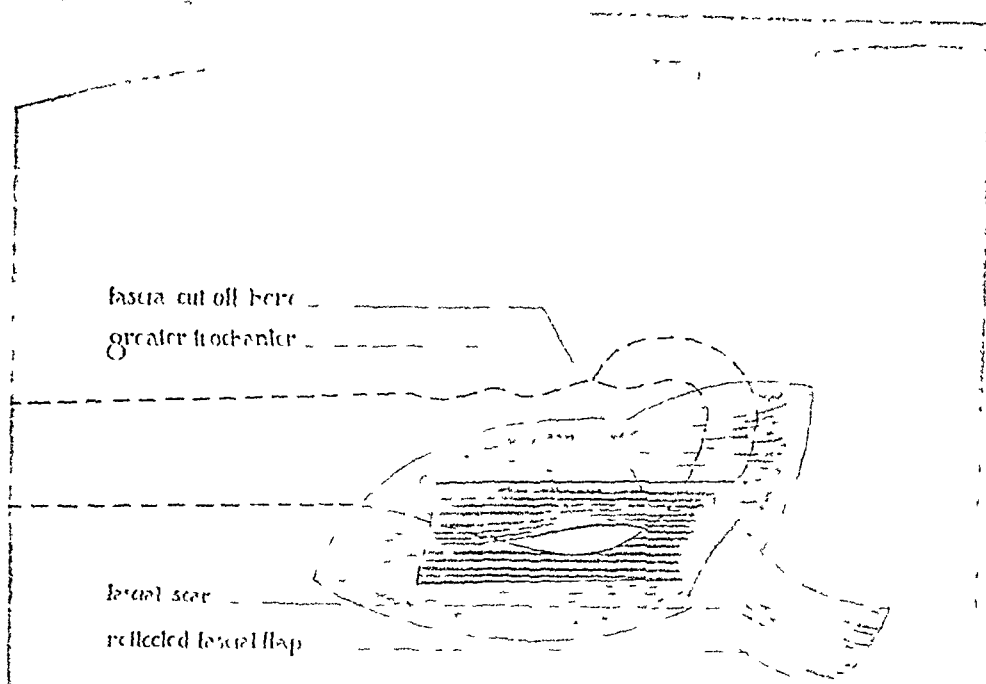
greater trochanter
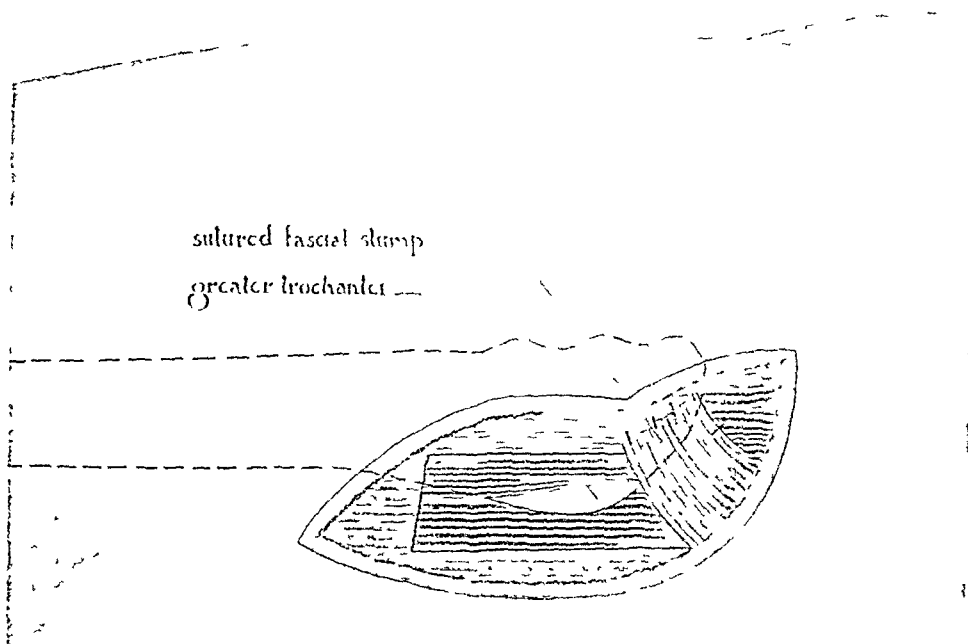
FIG. 4

FIG. 3. The fascia between the parallel incisions is dissected up and freed from the greater trochanter, thus also freeing the distal end of the tensor fascia femoris. The distal end of the flap is freed and turned back as shown. The distal two-thirds of flap cut off as shown by black line in above figure.

FIG. 4. The proximal one-third of the fascial strip is sutured anterior to and above greater trochanter to fascia of anterolateral surface of thigh. This absolutely prevents any further snapping of the hip from occurring. Defect in fascia is not closed.

mental surgeon who saw him thought that he had a possible hip dislocation and on admission to the hospital it had that appearance. X-ray was negative. There was no limitation of motion and no swelling, but he was unable to stand straight because of pain in the leg. The injured leg appeared shorter than its mate but was the same length on measurement. There was no popping. The patient was treated by bed rest. Four weeks later a slight snapping appeared in the left hip and gradually became worse with associated anterolateral thigh pain which was referred to the knee. This patient was badly disabled and unable to perform even the lightest military duty. At operation there was a thick iliotibial scar which was almost like cutting cartilage. Campbell's repair was done and the operation was a success. The patient is slowly recovering as he had a great amount of atrophy of the left thigh. No snapping or pain are present.

CASE III. M. C. K., age twenty-seven, injured his back and right hip during a blackout when an army truck turned over. X-ray was negative. All physical findings were negative on admission. Twenty days after the injury he began to have snapping of the right hip. This grew worse since walking was started and there was pain in the region of the trochanter. This patient has been advised to have surgical correction but as yet it has not been done.

CASE IV. P. V., age twenty-one, began to have snapping of the right hip four months ago, but pain did not appear until six weeks ago. There was snapping and pain in the anterolateral surface of thigh, greater trochanter and knee. At times he feels that the right hip is shorter than the left. He is beginning to have painless snap in the left hip also. There is no definite history of injury but it may have been due to a minor injury he received on march four months previously. Physical examination showed definite snap which is both palpable and visible on the right hip and a slight snap over the left. X-rays were negative for both hips. He was advised to have an operation on the right hip but refused.

CASE V. L. D. C., age eighteen, a male, experienced snapping of the right hip with every step which began three months ago. It was painless. No progress has been in this condition since the first month of onset. No definite history of injury was given. The snapping band

is easily felt over the trochanter. X-rays were negative. Legs are of equal length and there is no atrophy. As there was no pain, the pathology was explained and an operation was not advised. The patient was satisfied and he has had no further trouble with the hip.

CASE VI. L. P. M., age twenty-two, began to notice a popping of hip in the region of the greater trochanter whenever he took a step in winter of 1940. He had no pain. He felt the pop but did not hear it. There was no history of recent injury; however, in 1938, while playing football he received an indefinite hip injury which partially incapacitated him for two weeks. There was definite snapping on extension and flexion of hip. The thickened band can easily be rolled between the fingers of the examining hand. Legs are of equal length and there is no atrophy of thigh muscles. X-ray is negative. As there is no pain, an operation will not be advised. The patient, who is in the service, returned to duty.

In the above cases I have given only the most important points and have tried to touch only the high spots of each case. Two patients have been operated upon successfully, one awaits operation, another was advised to have surgery but refused, and in two no operation was indicated.

SUMMARY

Six cases of snapping hip are reported. The etiology, symptoms, physical findings, diagnosis, differential diagnosis, and operative technic are described. The results are given.

REFERENCES

1. CAMPBELL, WILLIS C. Operative Orthopedics. Pp. 839–840 St. Louis, C. V. Mosby Co.
2. DaCOSTA, J. C. Modern Surgery. Ed. 9, p. 701. Philadelphia, 1925. W. B. Saunders Co.
3. DICKINSON, ARTHUR M. Bilateral snapping hip. Am. J. Surg., 6: 97, 1929.
4. LOVETT, R. M. Orthopedic Surgery. In Civil Life. Keen's Surgery, 7: 590. Philadelphia, 1921. W. B. Saunders Co.
5. MAYER, L. Snapping hip. Surg., Gynec. & Obst., 29: 425, 1919.
6. PARSONS, E. B. The snapping hip. Texas State J. Med., 26: 361, 1930.
7. PRUITT, M. C. Snapping hip. J. M. A. Georgia, 10: 25, 1920.

SUBASTRAGALOID ARTHRODESIS FOR THE OS CALCIS TYPE OF FLAT FOOT*

DARRELL G. LEAVITT, M.D.

Diplomate of the American Board of Orthopedic Surgery

SEATTLE, WASHINGTON

IT will be necessary for the average orthopedic surgeon to alter to a certain degree his concepts of surgical indications in the treatment of painful or symptomatic flat feet in order to appreciate fully the unusual satisfaction to be realized from applying the operation of subastragaloid arthrodesis where it is specifically indicated. After several years of critical observation of postoperative surgical results in the treatment of various types of decompensated or painful flat feet, the author's opinion of the various operative procedures has been amplified especially as to where certain operative procedures were best applied, and where these various operative procedures yielded inferior results. After analyzing these results following surgery in the treatment of flat feet for sometime, it chanced during the examination of a child with a severe degree of flat foot that the author noted a marked valgus of the heels, which is considered to be so commonly associated with malposture of the feet, if not flat feet. It was observed that if the rear foot was held manually in the correct position; in other words if the valgus of the os calcis was manually prevented, when the child bore weight on the foot, the depression of the longitudinal arch with prominence of the structures on the medial aspect of the foot, either the scaphoid or head of the astragalus, or both, did not appear.

The thought then occurred that if by some operative procedure the deformity of the rear foot could be prevented, why would it not be possible to maintain the height of the longitudinal arch and prevent the commonly seen depression of the structures in the region of the medial foot, and the inward bulging of these same structures? Other patients were observed and examined with this thought in mind until it seemed convincing that such correction of and prevention of deformity would result from successful subastragaloid or subtalar arthrodesis. To date, the author has in the last four years performed, or has supervised performance on his service at the Children's Orthopedic Hospital in Seattle of about eight bilateral operations in which subtalar arthrodesis has been done, occasionally with simultaneous excision of a prominent accessory scaphoid bone, or subsequent excision of a remaining prominent or accessory scaphoid. The results from surgery in this class of patients has been the most uniformly satisfactory series of surgical results in flat feet experienced to date. In practically every case the result has been good or excellent. A few patients have some minor disturbances with the feet, but no serious return of deformity, while some of the feet have entirely normal appearance and apparent function. One of the first patients so treated has been able to walk as much as thirteen miles per day in the mountains in the summertime without difficulty. Reconstruction of the ligament holding the peroneus longus tendon in its groove was required on one side after the arthrodesis, however, in this first case.

What is the os calcis type of flat foot? Obviously, this condition is nothing new. However, if one will study the individual characteristics of the patient with foot complaints one will notice that in some of

these patients the valgus of the os calcis is a definite and important part of the deformity of the foot. Regardless of any

prominent on the medial aspect of the foot, and if the foot is flexible, even more commonly the scaphoid bone also in

FIG. 1. E B A, preoperative and B, postoperative standing photographs. Note improvement in foot posture. Retenaculum peroneus longus reconstructed from a strip of the Achilles tendon, left.

complex description of the mechanism and development of depression of the longitudinal arch of the foot in the weight-bearing position, it has become evident that one of the primary, if not the primary and initiating factor in the depression of the longitudinal arch in this type and the associated deformities of the flat foot lies in the occurrence of valgus of the os calcis when weight is borne. If the os calcis by weight bearing tilts into eversion or valgus, the head of the astragalus is permitted to gravitate medially in the horizontal plane, and having started in this direction undoubtedly soon, if not simultaneously, becomes plantar flexed in relation to the rear foot. The head of the astragalus then becomes very frequently

addition to becoming depressed plantarward bulges on the medial aspect of the foot. How much actual abduction of the forefoot also in the horizontal plane occurs through the midtarsal region is very difficult to determine, but in a flexible foot this probably is an appreciable entity, since the forefoot in standing or in a certain portion of the gait is fixed.

Practical application of subastragaloid or subtalar arthrodesis has proved that where bony contour and anatomic size of the foot bones are normal, fixation of the rear foot in the neutral or straight position will prevent rotatory deformity of the talus, as well as the leg above, in the horizontal plane, and equinous or plantar flexion deformity of this bone. (Rather,

if the shape of the bones of the feet is within normal limits, subtalar arthrodesis will prevent the bulging of the head of the

sory scaphoid bone, although the degree of protrusion of these structures on weight-bearing may be markedly decreased, it
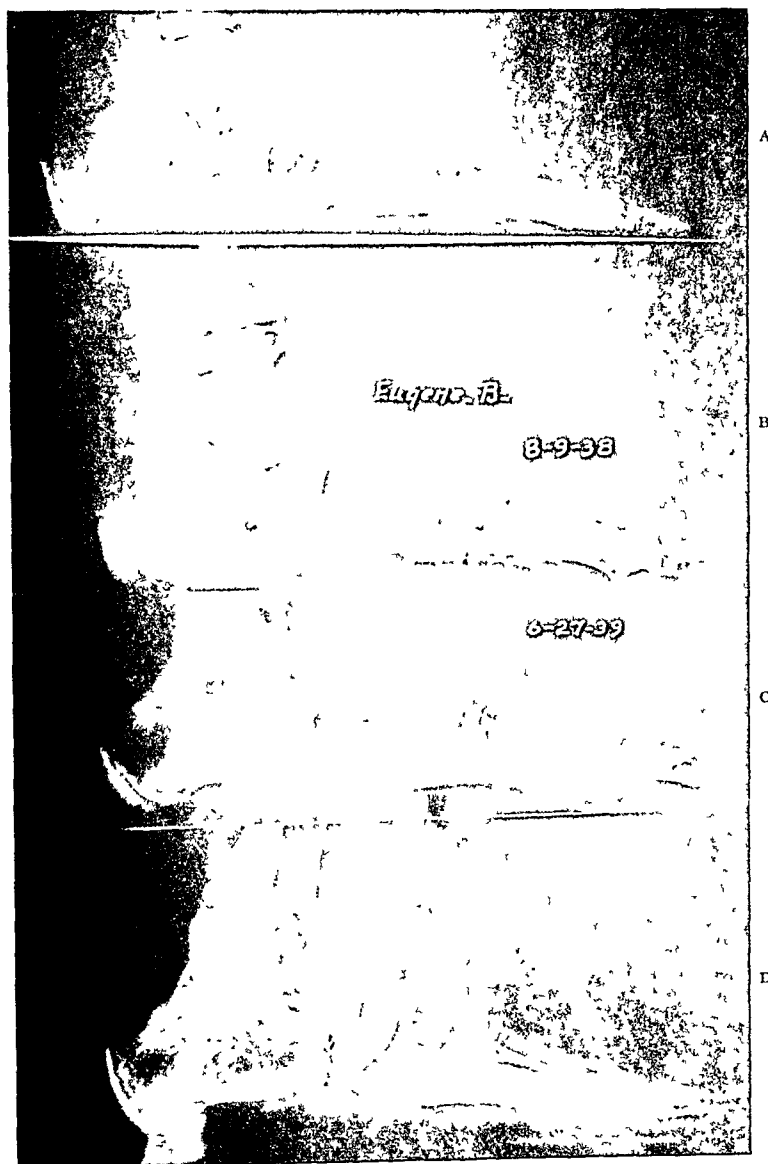
FIG. 2. E. B. A and B, preoperative; and C and D, postoperative standing radiographs, lateral views. Note subastragaloid arthrodesis, improved longitudinal arches.

astragalus medially, and also the prominence of the scaphoid on the inner aspect of the foot and concomitantly also prevents depression of the longitudinal arch in the weight-bearing position.) If, however, there is a congenital prominence and deformity of the neck or head of the astragalus, and a congenitally prominent enlarged or acces-

will be found necessary especially in the cases of the scaphoid protrusion to excise this fragment at the time or at a subsequent date. This is for the purpose of removing the prominence from the shoe irritation. In other words, not all feet in which scaphoid bone is very prominent, or in which an accessory scaphoid bone exists,
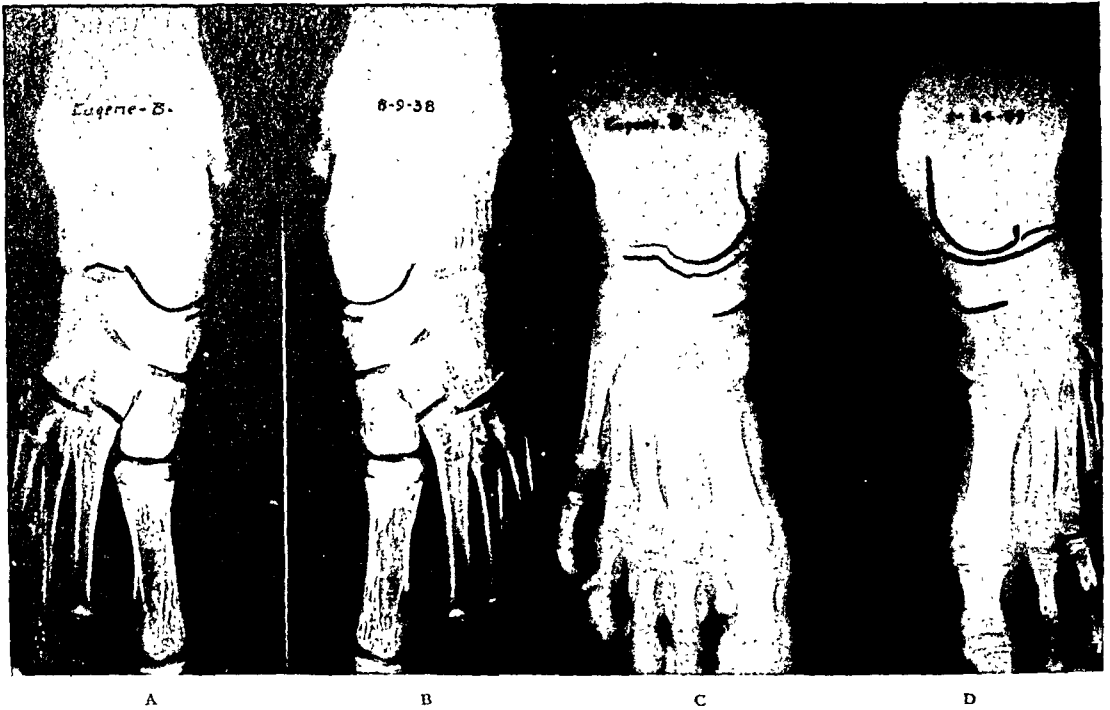
FIG. 3. E. B. A and B, preoperative and C and D, postoperative anteroposterior radiographs standing. Note some decrease in medial prominence of head of astragalus and scaphoid, right and left postoperatively.
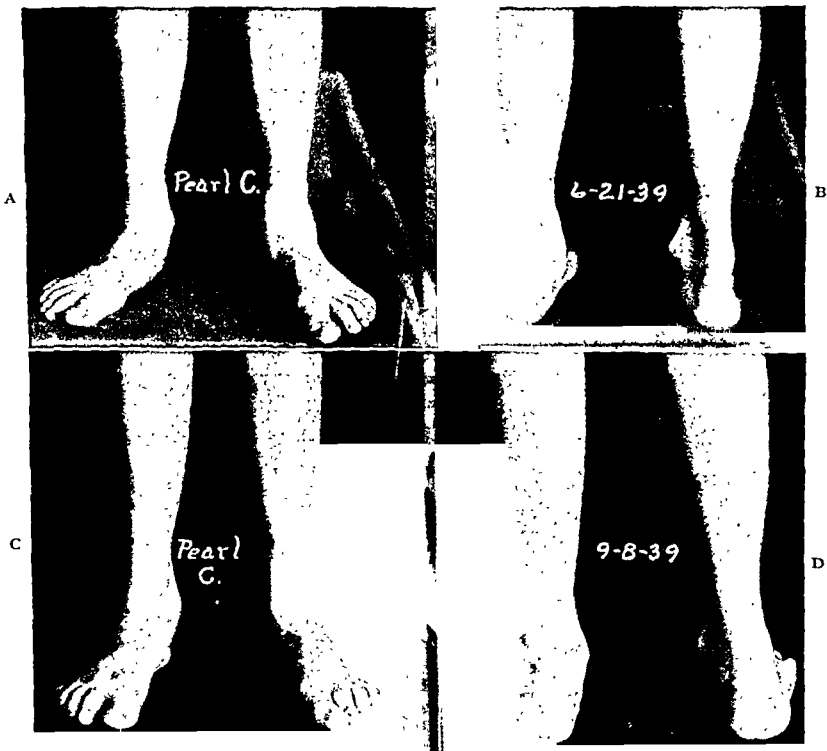
FIG. 4. P. C. A and B, preoperative and C and D, postoperative standing photographs. Note improved foot posture and increased height of longitudinal arches. Slight valgus of os calces apparent postoperatively.

are by any means flat feet, nor are they necessarily accompanied by symptoms of foot strain, but local pressure irritation may be the only symptom existing.

can be obtained in certain types of flexible flat feet by transference of the tibialis anticus tendon posteriorly, or possibly by other soft tissue surgery; but the
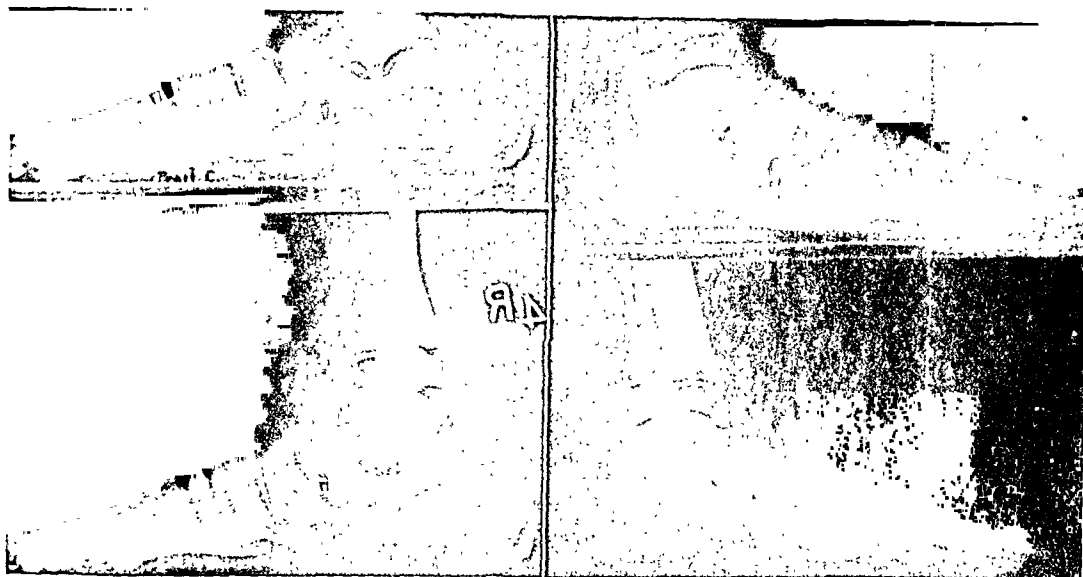
Fig. 5. P. C. Preoperative and postoperative lateral standing radiographs, postoperative showing sub-astragaloid arthrodesis and improved position of astragalus with higher longitudinal arch.
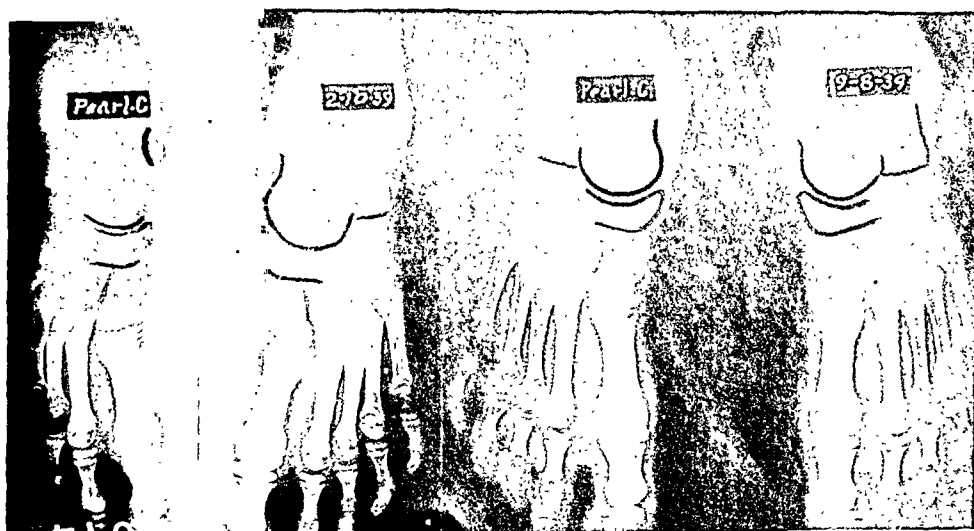
Fig. 6. P. C. Preoperative and postoperative standing anteroposterior radiographs, some change suggested less definite than lateral views.

A good deal is said and written about the inadvisability of disturbing the bony architecture of the foot or of removing the mobility of the foot joints when soft tissue work can be substituted. It is conceded that many excellent results

argument that interference with the joints of the foot is a factor of such importance that it should be avoided is, and has been proved by the results in the os calcis type or flat foot to be fallacious. The lack of instability in the rear foot is insurance

against repeated foot strain under function in which the foot has proved itself to be incompetent in the first place. Postopera- supination of the forefoot in the presence of fusion in the talocalcaneal or subtalar joint. That they do not have entirely
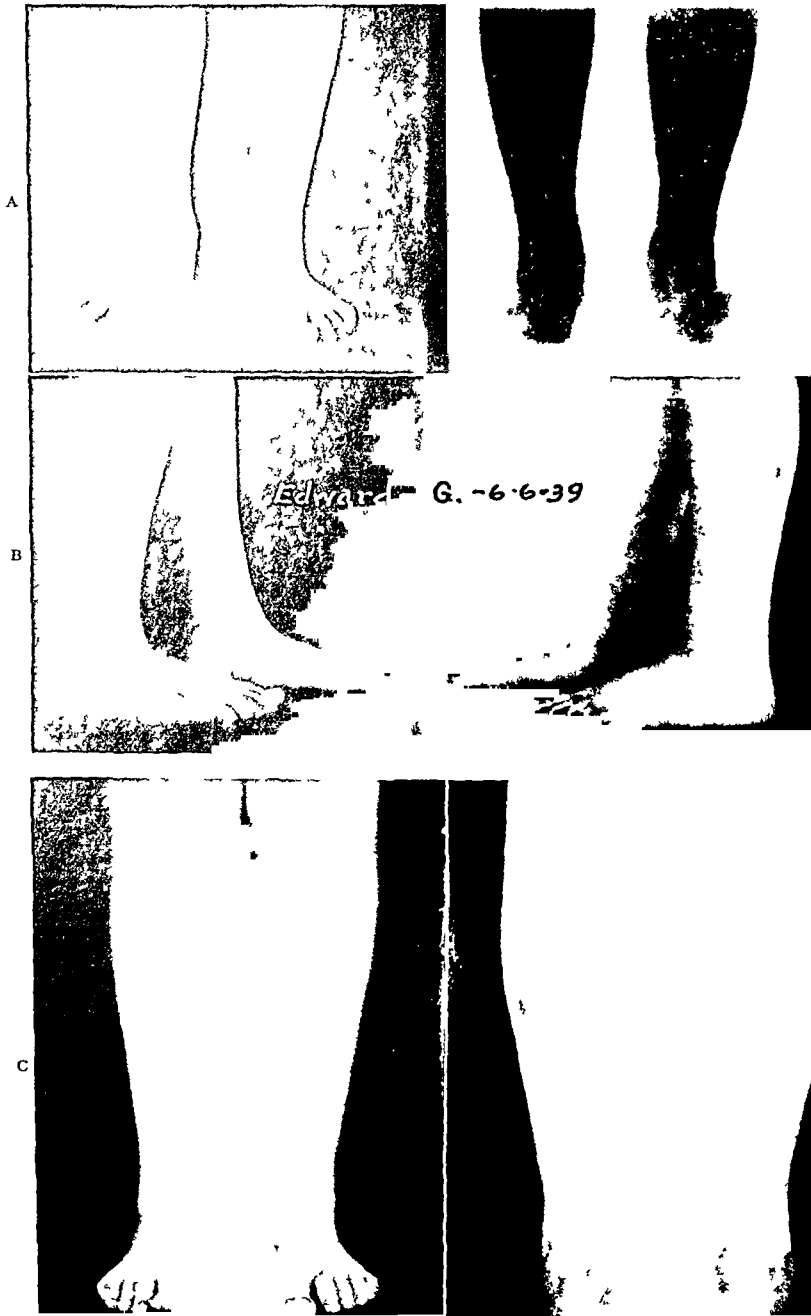
FIG 7. E G A and B, preoperative standing photographs, C and D, postoperative standing photographs.

tive examination of the patients reveals that they have maintained a large percentage of the passive pronation and normal or flexible excursion of pronation and supination in the anterior portion of the foot is a condition to cause probable
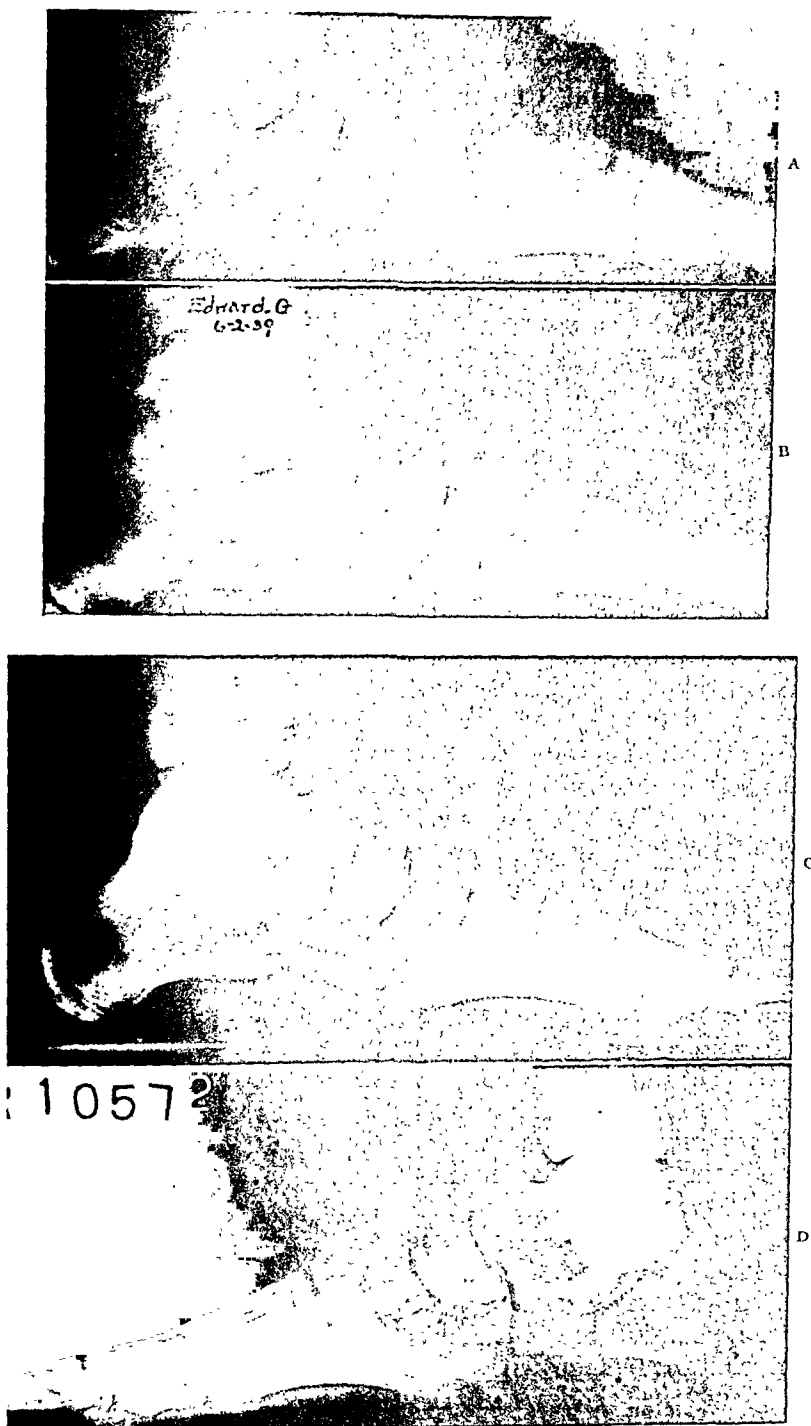
Fig. 8. E. G. A and B, preoperative standing lateral radiographs; C and D, postoperative lateral standing radiographs. Note resultant increase in longitudinal arch and higher position of scaphoids and head of the astragali.

rejoicing in years to come. Until the present time the author has not performed this operation for the painful os calcis type of static flat foot in adults, but its possibilities are not restricted to this type of flat feet in children.

The operative procedure is certainly not new. The recognition of the type of symptomatic flat foot in which valgus or eversion of the os calcis is the initiating deformity is comparatively simple, if it is considered. The operation itself because of the limited exposure is not easier than a triple arthrodesis, and at first may seem more difficult to the operator. Ordinary care will prevent appreciable injuring of the midtarsal joints. It is unnecessary to remove a wedge as the resting posture of these feet usually approximates normal. If the bony surfaces are deeply chipped and held in the neutral position until fusion occurs, subsequent deformity should be prevented. After the first few operations it was noted that the relative position of the head of the astragalus with reference to the anterior tubercle of the os calcis was better preserved in the postoperative non weight bearing radiograph when the foot was markedly inverted and adducted with some plantar flexion; but since fixed varus of the posterior foot is a very undesired complication to subtalar arthrodesis, the author has habitually returned the foot to the walking position with the heel neutral in approximately three weeks. Two or more months of plaster immobilization with a subsequent period of longitudinal arch support were carried out. While preoperative standing radiographs made from the lateral aspect have shown the head of the astragalus to lie at a much lower plane with relative overriding of the shadow of the anterior tubercle of the os calcis, postoperative radiographs have demonstrated a variable but uniformly higher plane of the head of the astragalus; and the long arch also is apparently higher. In the anteroposterior standing radiographs there is

suggested, but less clearly, improvement in the outward swing or abduction of the forefoot, especially through the midtarsal joints. The most visible evidence of correction in the postoperative photographs, which are certainly variable in judging postoperative results, show decrease or absence of the valgus of the heel and marked tendency for recession of the prominent structures in the medial aspect of the foot, particularly in the patients in whom accessory scaphoid prominences are not present. In one instance the author attempted to pin the neck of the astragalus to the os calcis in the correct position by a Kirschner wire, but this first trial was apparently not anymore successful than on the side where plaster alone was used, although the patient had definitely less discomfort in this foot postoperatively than in the other side.

The following preoperative and postoperative photographs are presented to demonstrate some of these patients without special regard for choosing those considered most satisfactory. (Figs. 1 to 8.)

CONCLUSIONS

1. The os calcis type of flat foot may be defined as a postural deformity of the foot in which apparently valgus or eversion of the rear foot initiates and is responsible for the subsequent deformities associated with depression of the longitudinal arch.

2. Functional deformity of the os calcis type of flat foot without abnormal congenital protrusion or deformity of the head of the astragalus and especially of the scaphoid can be prevented by subtalar arthrodesis.

3. Symptoms of decompensation or foot strain accompanying this type of flat foot can best and most satisfactorily and permanently be corrected by subtalar arthrodesis.

4. It is sometimes necessary to remove the abnormal prominence of the scaphoid bone.

WELL THIGH TRACTION IN THE TREATMENT OF INTERTROCHANTERIC FRACTURES

IRVIN STEIN, M.D. AND RUBIN M. LEWIS, M.D.

Chief Orthopedic Surgeon, Philadelphia Assistant Orthopedic Surgeon, Philadelphia
General Hospital General Hospital

PHILADELPHIA, PENNSYLVANIA

THE problem of reduction of intertrochanteric fractures has always been a serious one. Maintenance of reduction has also presented difficulties. Because most of the patients admitted to the Orthopedic Service of the Philadelphia General Hospital are aged and debilitated and over seventy years of age, hospital care and extended periods of hospitalization aggravate the basic critical problem.

In an attempt to solve the problem as a whole, we considered it necessary to accomplish the following: (1) Adequate reduction; (2) suitable fixation; (3) simplified nursing care; (4) simple operative procedure; (5) freedom of hip and knee motions, and (6) low cost apparatus.

Reviewed and considered were the merits of such existing methods as: (1) Open operation; (2) pin and screw fixation, and (3) well leg traction and fixation—Roger Anderson well leg splint and well leg casts with struts.

operation, for example, in the type of cases we are called upon to treat, is too shocking

FIG. 1. Illustration showing the scheme of the apparatus and an analysis of the forces.

a procedure to warrant serious consideration. It was, therefore, dropped.

FIG. 2. Scale drawing of the apparatus.

Definite limitations appeared in the survey of these available technics. Open

Pin and screw fixation may be suitable in some cases. In many of our patients, how-

ever, the site of the fracture and the demineralization of the bone do not make the use of this method feasible.
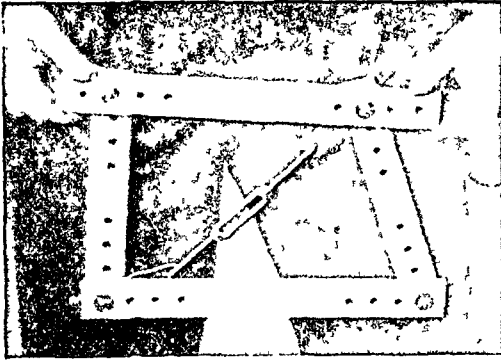
FIG. 3. Photograph of apparatus.

The high cost of a large number of apparatus must not be overlooked when it is considered that there are about ten or twelve of these cases in our wards at one time. At least a dozen pieces of this apparatus is therefore required. To avoid this initial expense, the solution is to be found in an inexpensive apparatus, but one which is effective and efficient.

The Roger Anderson well leg traction apparatus provides a very satisfactory method for control of these cases. Unfortunately, here, too, there are drawbacks. It

"glue up." Although it is not an expensive piece of equipment, if one case alone is involved, a heavy outlay of money is called
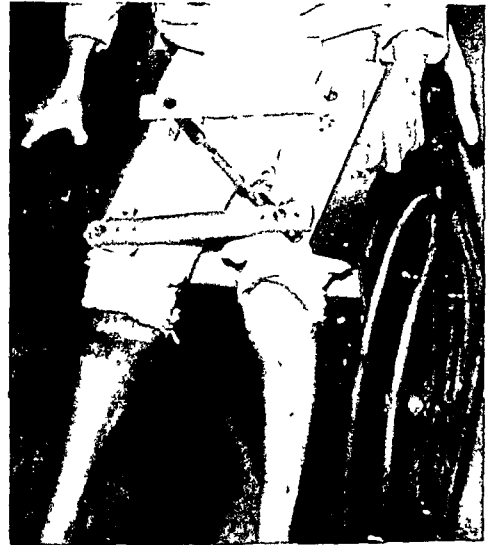
FIG. 4. Photograph of apparatus as applied to patient.

for when ten or twelve apparatus are needed.

In discussing this problem with another of the orthopedic chiefs at the hospital, we learned that Dr. J. R. Moore used the Anderson well leg apparatus with a pin part way through the femur rather than

FIG. 5.

FIG. 6.

FIGS. 5 and 6. B. Z. Intertrochanteric fracture before and after application of apparatus.

meets all but the last two requirements in our specifications. Although the Anderson apparatus allows motion of the hip joints, the knees are fixed and have a tendency to

through the tibia on the fractured limb. In this way traction on the ligaments of the knee was eliminated and a subsequent relaxed joint obviated.

After further study of the whole picture a solution was presumed whereby both knees could be left free and traction and pressure applied through pins at the lower ends of the femora. To accomplish this a mechanism to provide the necessary forces and fixation was devised.

The materials used are as follows: two cross bars—Maple, birch or waterproof plywood; two longitudinal bars; four machine screws and nuts; four washers; one turnbuckle and two pieces of ⅜ inch by 4 inches—18 gauge strap iron.

TECHNIC

1. Local anesthesia is preferred. Steinman pins sufficiently long to extend one inch to one and a half inches beyond the skin margins are used. These are inserted approximately one inch above the abductor tubercle of the femur. They are placed in the coronal plane perpendicular to the shaft.

2. A felt pad, three inches wide, one-half inch thick and long enough to almost reach the pin on both sides of the thigh is placed just proximal to the superior border of the patella. This in turn is held in place by cotton sheeting wrapped around the thigh. Except for this padding, the plaster is applied directly to the skin.

3. The casts are applied from the knee to the groin. Care must be taken, however, to place the plaster high on the lateral surface of the fractured thigh.

FIG. 7. P. H. Intertrochanteric fracture.

4. Place the longitudinal bar (Fig. 1) on the anterior surface of the well thigh cast in

FIG. 8. P. H. Intertrochanteric fracture following application of apparatus.

the sagittal plane. Embody this link in the plaster and fix it to the pin.

5. Place the longitudinal bar (Fig. 2) on the anterior surface of the fractured thigh cast fifteen degrees lateral to the sagittal

plane and fix it to the cast in similar fashion. This is to provide the internal rotation necessary to align the fragments.

6. The patient is returned to the ward and the plaster is allowed to dry.

7. The turnbuckle assembly is placed from the proximal bolt on the fractured thigh to the distal bolt on the well thigh. The cross links are placed and the nuts are screwed down on the bolts. It is necessary to rotate internally the fractured thigh to place the crossbars. This can be accomplished with little pain if gradual firm traction is used while internally rotating.

8. The turnbuckle is tightened and a roentgenogram is made. If reduction is not complete, additional tension is applied through the tightening of the turnbuckle.

SUMMARY

A discussion of the problems in the treatment of intertrochanteric fractures of the femur is presented.

Specifications for a suitable apparatus are outlined; a description of the mechanism and design are shown; the technic of application is given and the fractures and their reduction are shown by the roentgenograms.

This paper is a preliminary report and end results are not indicated.

※※※

ANY phalangeal joint may be dislocated backward, forward or laterally. Reduction is by traction and bending the finger in the direction opposite to the dislocation. Occasionally, repair of the capsule is necessary to prevent recurrence of the displacement.

From "A Manual of the Treatment of Fractures" by John A. Caldwell (Charles C. Thomas).

THE SYNDROME OF THE FIXED OMENTUM*

GLENN ASHWORTH, M.D.

On the General Surgery Staff, Myers Clinic Hospital
PHILIPPI, WEST VIRGINIA

ATTACHMENT-FIXATION of the great omentum produces a guy-rope action of this structure upon the stomach, duodenum, transverse colon and diaphragm. The tension exerted by this relationship induces the signs and symptoms which characterize a separate and distinct clinical entity. I refer to this entity as "The Syndrome of the Fixed Omentum."

ETIOLOGY

The normally free margin of the great omentum can become fixed under a large number of conditions, the principal conditions being: (1) To incisional scars; (2) to inflammatory masses; (3) to raw peritoneal surfaces, and (4) herniation and incarceration into hernial sacs.

The extent of fixation attachment varies considerably in regard to the total surface of the omentum involved and in regard to the region of the omentum which becomes attached. This varies from attachment-fixation of the "total" surface, in tubercular peritonitis, to that of a single taut, cord-like strand of fibrous omentum, the violin-string adhesive band.

Traction of this fixed point on the walls of the stomach, duodenum, transverse colon and diaphragm induces the symptom-complex. The severity of the symptoms of the syndrome depends upon the direction of the traction and upon the resting tension of the attachment-fixation.

I have seen a strand of omentum fixed to the right ovary after partial oophorectomy; fixed to the entire posterior surface or fundus of the uterus; incarceration-fixed in inguinal, femoral, umbilical, lumbar or incisional hernias, to the sigmoid colon, the cecum, a McBurney incision, a midline incision or the various other regional incisions, fixed to an appendix, to a Meckel's diverticulum or diverticulum of the sigmoid colon, and to malignant growths.

PHYSIOLOGY: PRODUCTION OF GASTRIC PAIN

The innervation of the stomach consists of the pressor and depressor fibers of the autonomic nervous system. The sympathetic innervation transmits impulses to the celiac plexus, thence to the seventh, eighth and ninth thoracic cord segments and from there to the midbrain. The receptors are not in the cerebral sensorium and, therefore, do not record pain. These fibers transmit a reflex stimulus to the muscles and to the cutaneous structures supplied by these dorsal cord segments, with resultant reflex muscular rigidity and cutaneous hyperesthesia limited to the epigastric region. The sympathetic fibers record fulness and discomfort but do not record pain.

The parasympathetic fibers convey impulses to the sensorium of the brain and can, therefore, record painful sensations. The stomach records sensitively the fact that it is full; it is relatively insensitive to heat, to cold and to touch. Increased tension of the gastric wall induces pain, nausea and vomiting. During an operation upon the stomach, under local anesthesia, the surgeon can handle the stomach without producing untoward symptoms to the patient. Traction upon the stomach

induces pain, nausea, vomiting, brady-cardia and general vagotonia.

An increased tension of the gastric wall sufficient to induce pain results: (1) From distention of the viscus from within (pulsion tension); (2) from spasticity of the muscular wall of the viscus (intrinsic tension), and (3) from traction on the viscus from without (traction tension).

Distention of the stomach beyond the limits of "active" physiological relaxation, as in the late stages of acute gastric dilatation, distention of the leather-bottle and hour-glass stomachs, and distention of the stomach when chronic diffuse gastritis or when diffuse malignant infiltration is present, all represent types of pulsion-tension. Pain during the filling phase of gastric digestion characterizes these conditions.

Spasticity of the musculature of the stomach wall results from localized inflammatory infiltration, as in ulcer or eroded malignancy, from acute hyper-peristalsis of the stomach, as illustrated by sudden obstruction of the pylorus, duodenum or jejunum, and by the resistance to peristalsis of polypoid growths of the stomach. Pain during the emptying phase of gastric digestion characterizes these lesions.

Traction on the gastric wall results from ptosis of the transverse colon when this organ is abnormally distended with fluid; from fixation-attachment, either of the wall of the stomach to a stationary tissue or of the appendages of the stomach to a stationary tissue. This applies especially to the omentum, the attachment-fixation of which is our chief concern in this communication.

ANATOMY

The stomach lies suspended in the upper abdomen by the anlages of the dorsal and ventral mesogastrii. The ventral mesogastrium, or lesser omentum, incorporates the structures of the liver. Its suspensory function of the stomach is brought about by the transmission of its traction to the suspensory ligaments of the liver, which attach to the diaphragm and to the anterior and posterior abdominal walls. The lesser curvature of the stomach is suspended, therefore, either directly or indirectly from the diaphragm by the meso-esophagus, the hepatogastric ligament, the flaccid lesser omentum and the hepatoduodenal ligament, which arises from the ventral mesoduodenum. The ventral mesoduodenum overlies only the proximal duodenum and it bounds the foramen of Winslow anteriorly. Below the foramen of Winslow the duodenum is attached, by fusion, to the parietal abdominal wall overlying the right kidney and the lateral margin of the diaphragm.

The great omentum develops from the dorsal mesogastrium by an overgrowth, which is out of all proportions to the demands of gastric rotation on this structure. The anterior and posterior layers of this dorsal mesogastrium fuse together and this structure subsequently fuses with the transverse mesocolon, suspending the transverse colon from the greater curvature of the stomach. Fusion of the great omentum extends laterally to the diaphragm, beyond the flexures of the colon, and it aids in the suspension of the colon at the flexures.

Traction on the great omentum, therefore, is transmitted to the greater curvature of the stomach and of the duodenum, to the walls of the stomach and of the duodenum, to the lesser curvature of these structures, to the lesser omentum, to the liver and, from thence, to the diaphragm. The traction is transmitted to the transverse colon and from this structure to the diaphragm by way of the suspensory ligaments at the flexures of the colon.

The greater curvature of the stomach occupies a station in the abdomen considerably lower when the stomach is empty than when it is full. When the lumen of a distended stomach forms an oval ten inches in circumference, its diameter is approximately three inches. This section of the stomach when collapsed would form two flat, opposed surfaces five inches broad. At

this cross-section of the stomach, therefore, the attached great omentum would assume a station two inches lower in the abdomen when the stomach is empty than when it is distended. It can be seen that, should the free margin of the omentum become attached in the lower abdomen when the viscus is empty, any attempt to elevate the greater curvature of the stomach, either by distending the organ with food or by stretching would result in a guy-rope action of the great omentum on the stomach. This guy-rope action induces an increased tension on the structures of the stomach wall, which is productive of a painful dyspepsia of the "traction-tension" variety.

This tension is accentuated by any further separation of the fixation points of the stomach, that is, the fixed lesser omentum and the fixed region of the great omentum. Stretching, lifting, deep expiration or pressure exerted transversely across the abdomen, above the level of the omental fixation, will accentuate the tension. The tension is reduced, the symptoms being alleviated, by the approximation of the fixation points of the stomach, as in the act of "doubling-up," vomiting and deep inspiration.

CASE HISTORY

The syndrome of the fixed omentum presents the following phenomena:

1. These patients complain of a full-stomach, painful dyspepsia; the pain occurs in the epigastrium.

2. They feel a traction-discomfort in the lower part of the abdomen when they stretch, as in reaching for an object upon a high shelf; the region of the traction is, usually, readily located.

3. They have frequent episodes of nausea and vomiting, immediately after eating. Vomiting relieves the pain and distress.

4. The traction-discomfort is frequently accompanied by epigastric pain and nausea. This is especially noticed when the patient lifts a heavy object or elevates the arms above the head, as in stretching.

5. Attacks of intestinal colic are common and a history of audible borborygmi is the rule.

6. The symptoms are absent when the stomach is empty, except when positive tension is constantly evident. They are relieved when the patient lies on his side and flexes the knees on the abdomen, when the food intake is limited to fluids or to small quantities of solids, or when the patient induces vomiting.

7. These patients are usually well nourished; they do not present the clinical picture of an organic lesion of the gastrointestinal tract, with the progressive onset of wasting and debility, without remission. These patients have remissions of symptoms and frequently go for considerable symptom-free periods by controlling their diet and habits. In this series of cases, the shortest history obtained was one of fifteen weeks; the longest history obtained was one of thirty-four years.

8. These patients frequently complain of compression of the chest, palpitation and pains in the chest. Deep inspiration relieves the condition, whereas, deep expiration accentuates it. This class of symptoms (cardiorespiratory) is noted principally in cases of positive tension.

9. A history of previous abdominal surgery, of the presence of hernia or of an abdominal crisis, in the recent or distant past, is usually obtained.

PHYSICAL EXAMINATION

The physical signs can be elicited by tests based upon the mechanical nature of the lesion.

When the recumbent patient is instructed to attempt to assume the sitting posture without using his arms, retraction upward of an abdominal escher is frequently observed. Lower abdominal eschars are frequently observed to retract to such an extent that the panniculus overhands the upper reaches of the eschar. An incarcerated omentum will occasionally reduce a scrotal hernia into the abdomen when this test is effected. Retraction of this variety can be felt as well as be observed.

When the patient stretches, as in reaching for an object upon a high shelf, the symptoms are induced or accentuated.

When the patient lifts a heavy object the symptoms are induced or accentuated.

Distending the stomach with food or drink induces the symptoms; stretching further accentuates the symptoms and "doubling-up" relieves them.

Deep expiration, while the patient "stretches," induces the heart and respiratory "compression" symptoms, especially in positive-tension cases. ·

Transverse compression of the abdominal wall above the fixation level induces *epigastric pain, nausea and lower abdominal traction discomfort.*

These patients are reasonably well nourished; they have a stable nervous background; and they accurately localize the pain in the epigastrium and the traction-discomfort in the lower abdomen.

X-rays taken of these patients are reported as "negative." A lateral x-ray of the stomach, when distended with barium, shows a tendency toward a fusiform outline instead of an oval outline. A flatness or plateau is frequently observed instead of the rounded dome of the diaphragm when the patient stretches during fluoroscopic examination and exhales.

Stasis in the right half of the colon is frequently noted, due to the compression of the transverse colon against the spine. I have no proven instance of a duodenal stasis due to pressure of this variety but such is possibly the case.

Auscultation demonstrates hyperperistalsis of the stomach, small bowel and the right half of the colon. This finding disappears after the patient has "doubled-up" for an interval.

DIFFERENTIAL DIAGNOSIS

This syndrome must be differentiated from the full stomach dyspepsias, which conditions include: stenosis of the pylorus of the stomach, partial obstruction of the small or large bowel, diffuse gastritis, diffuse malignant infiltration, leather-bottle and hour-glass stomachs, gallbladder

disease, chronic passive congestion and visceroptosis. By the same token, this syndrome must be recognized when it accompanies other intra-abdominal lesions. This is exemplified by the case which presents an incarcerated omentum in a hernia sac; transfixation and transplantation of the sac without releasing the omentum serves only to make permanent the symptom-producing fixation.

This syndrome frequently accompanies peptic ulcers. This group of patients attempts to keep small amounts of food in the stomach by frequent small meals. Whether or not the tension produced on *the gastric wall by the omental-fixation* contributes to the production of peptic ulcers, is a matter of conjecture.

TREATMENT OF THE FIXED OMENTUM

The resting-tension of the fixation determines the ultimate choice between nonsurgical and surgical management of the individual patient. The degrees of resting-tension of the fixed omentum are divisible into three clinical groups: (1) Those with *lax-tension*, in which cases conservative management is all that is needed; (2) those with *taut-tension*, in which cases conservative treatment is given a trial, with the necessity of surgical intervention and imminent probability; and (3) those with *positive-tension*, in which cases surgical release of the omental-fixation is the only means of relief.

Discreet re-peritonization of all denuded surfaces and the correction of the co-existent lesion (such as hernias, malignancy or inflammatory lesion) are essential duties of the surgeon for proper conclusion of the procedure.

Conservative management is applicable to groups 1 and 2, the objective being to reduce the traction-tension of and to maintain as much laxity of the omentum as is possible. The following instructions are given to the patient: (1) Eat smaller quantities of food at each meal; eat more frequently; (2) avoid roughage foods (liquids and soft foods rapidly leave the stomach and cause less distress); (3)

maintain a soft stool by means of mineral oil, psyllium seed or similar medication; (4) during distressing intervals limit food intake to liquids; (5) dietary instructions, insuring an optimal vitamin intake and, if indicated, weight reduction; (6) when it is possible to elevate the abdominal parietes in the region of the attachment-fixation and by so doing relax the resting-tension of the attachment-fixation, a properly constructed corset should be fitted to the patient; (7) when necessary, either to prepare the patient nutritionally for surgical release of the omentum or to build up a debilitated patient, the patient is instructed to lie on the right side with the knees flexed on the chest for forty-five minutes to one hour after each meal.

Surgical management is applicable to groups 2 and 3, the objective being to release the omentum, re-establishing its normal status of free mobility in the abdominal cavity, and to remove the traction-tension from the structures of the stomach wall.

In releasing the fixed omentum I have abandoned the sharp dissection in the white plane of cleavage. I amputate the omentum in such a manner that a minimal amount of omental tissue is left at the site of the fixation. This residual omental tissue presents a smooth peritoneal surface, its demand for a blood supply is small and the graft is already successfully established. I suspect that considerable of the fat of this resident segment eventually undergoes absorption and that subsequently only a smooth eschar remains.

The vessels of both the resident segment and of the proximal, released margin are transfixed with very fine sutures, care being taken to incorporate the minimal amount of tissue with each vessel. The margin is then "rolled" upon itself, and secured by means of a fine continuous suture, in such a manner that no raw surface of the omentum remains exposed.

CASE REPORT

CASE I. Miss Helen P. complained of fulness, gas, nausea and vomiting after meals;

upper abdominal pain, and lower abdominal traction discomfort after meals. These symptoms had been progressive since a bilateral salpingectomy, left oophorectomy and right partial oophorectomy had been performed fourteen months before. She had suffered an acute, lower, right abdominal pain, of extreme severity, of a colicky nature, accompanied by severe vomiting, for six hours before admission.

The patient stood with the chest flexed on the abdomen. Attempts to straighten up intensified the pain, nausea and colic. When the patient reclined and doubled up the acute symptoms partially subsided. On auscultation, vigorous peristaltic sounds, localized in maximum intensity in the right lower quadrant, were heard. The abdomen was soft. On bimanual examination, a mass was palpable in the region of the right pelvic adnexa. *Preoperative Diagnosis:* Omental fixation in the pelvis; acute ileal obstruction.

Operative findings were a cord-like, fibrous strand of omentum, attached to the remains of the right ovary. A loop of ileum was incarcerated beneath this fibrous strand. This strand was dissected carefully from the surface of the ovary and the incarcerated ileum liberated. The strand was removed by a partial amputation, of this region of the omentum, the cut margin of the omentum being rolled and sutured in such a manner that no raw surface presented. No further pathological condition was noted on careful examination. The patient's recovery was uncomplicated and she has had no untoward abdominal symptoms since.

CASE II. Mr. John M., Jr., age nineteen, stated that he had had attacks of epigastric pain, fulness, belching, nausea and vomiting following his meals, since early childhood. These attacks were related to the size of the meal eaten and tended to occur more readily and more severely when solids were eaten than when liquids were drunk. He had been treated for a number of years for pylorospasm without improvement. He was unable to obtain employment because lifting, driving an automobile or stretching induced his symptoms and disabled him.

On examination a large, congenital scrotal hernia was found on the right side. This hernia reduced itself when the patient laid recumbent and extended his arms over his head. At operation a firm, fibrous fixation-attachment of the omentum to the hernis sac was found. Release of the omentum and repair of the

hernia has rehabilitated this young man to full occupational activity, with no recurrence of his symptoms.

CASES III AND IV. Two patients presented a similar clinical picture, which proved to be due in each instance to compression of the transverse colon against the spinal column by the fixed omentum. Classical signs and symptoms of omental attachment-fixation were present in each case. In addition, each patient presented signs and symptoms of chronic, partial obstruction of the transverse colon. In each instance a barium meal almost completely stopped in the midregion of the transverse colon and a barium enema progressed normally to the midregion of the transverse colon but halted there as long as the patient remained recumbent. When the patient was turned to a right-sided Sims' position, the distress diminished and the column of barium passed readily to the cecum.

When these patients were operated upon, Mrs. H. A., Sr., presented an attachment-fixation of the omentum to the entire posterior surface of the uterus and to the sigmoid colon; Mr. D. W. presented a fibrous attachment-fixation of the omentum to a Battle's incision in the right lower abdomen. Careful abdominal explorations, in each instance, revealed no other pathologic process in evidence.

CASE V. Mrs. D. C., age forty-five, who weighed 262 pounds, presented a large, lower-abdominal, midline, postoperative ventral hernia. She had extreme omental fixation signs and symptoms, which were relieved by the tight application of a corset before arising each morning. At operation the abdominal cavity was entered to the left of the hernia sac and the fingers were passed upward and medially between the omentum and the anterior abdominal wall. Pressure was exerted on the anterior surface of the omentum and this structure was found to be taut and under considerable tension. By exerting pressure upon the anterior surface of the omentum wretching was repeatedly induced.

CASE VI. Mr. W. J. Eleven months prior to admission, this patient submitted to surgical repair of a right indirect inguinal hernia. Subsequent to this operation, he experienced an intensification of his original symptoms and, in addition, developed an ulcer dyspepsia. Examination revealed a complete clinical picture of omental fixation; x-rays demonstrating a duodenal ulcer were obtained. At operation the omentum was found to be firmly attached to the incisional eschar. The released omentum presented a black silk suture. The hernia sac had been transfixed and transplanted with the attached omentum incorporated in the suture. A satisfactory response to medical treatment of the ulcer made it possible for this patient to return to full occupational duty without symptoms or distress.

SUMMARY

1. The syndrome of omental attachment-fixation has been presented.

2. The production of the symptoms of this entity have been explained.

3. A division into clinical groups has been advocated and methods of management presented.

4. A method of amputating the omentum is presented.

5. The differential diagnosis of this syndrome from other lesions is discussed.

6. Case reports are presented to illustrate various types of the clinical manifestations of the syndrome.

7. Further conclusions follow these facts: (1) The syndrome is a very common one. (2) More discreet closure of abdominal incisions is necessary, to prevent attachment-fixation of the omentum, as is also the discreet re-peritonization of raw intra-abdominal surfaces. (3) The unwarranted usage of intra-abdominal drains, unnecessary suture material or other foreign bodies must be sharply delimited. (4) The differential diagnosis of this syndrome from other intra-abdominal lesions makes the proper diagnosis of intra-abdominal pathological conditions more accurate and more inclusive, and provides assurance of surgical relief to a new and considerable group of patients. (5) Distinction should be made as to whether or not this syndrome complicates other intra-abdominal lesions; for example, when fixation-attachment of the omentum accompanies an intra-abdominal lesion, such as hernia. (6) In the preoperative analysis of these cases full consideration must be given to the estimation of bowel segment fixation and to eschar compression of the ureter.

HERNIOPLASTIC OPERATIONS UPON THE RECTUM AND PELVIC FLOOR

CHANNING W. BARRETT, M.D.

Gynecologist, Wesley Memorial Hospital; Emeritus Fellow of the American Association of Obstetricians and Gynecologists

CHICAGO, ILLINOIS

WE cannot well discuss the above subject without considering the changes which eons of time wrought with three tail muscles to prepare the caudal end of the abdominal cavity to meet the extra demands of support in the upright position. These muscles have exchanged length, grace and agility, that were able to make a whiplash of the tail, for breadth and compactness and stability, that makes of the abdominal floor a bulwark of strength and yet a facile passage for the three important tracts which lead from the abdomen to the external world. A careful study has been made by tireless investigators in a series of animals as they represent the different degrees of the change to the upright position. The essentials of this change will be met for our present purpose if we can get a good understanding of the finished product. They are the pubococcygeus, the iliococcygeus and the ischiococcygeus, or coccygeus on each side blending into the levator ani muscles of the corresponding side. These cease to be individualists; they drop in part their coccygeal attachment, retain and amplify the circumference origin, and insert into the coccyx, into the median raphé between the coccyx and the anus, alongside the rectum and unite to the central tendon between the rectum and vagina, sending some fibers to the vagina and around the urethra. This makes then with the fascia above and below a broad complete musculofascial aponeurotic plate or griddle, which closes the lower wall of the abdomen— the pelvic floor—with tight clefts through which the tracts pass. In passage these tracts are grasped with snug fitting musculofibrous structures which decussate with the structures of the tubes, which so accommodate as to allow these tubes their functioning capacity, but which hold their walls in contact during periods of rest. The vast amount of work of adaptability over eons of time, carefully adjusted to furnish support and meet resistance and yet furnish easy passage to functioning processes would seem, if verified by the anatomist, physiologist and surgeon, to furnish an argument far too strong for the advocates of the prolapse of the rectum theory, namely, to take the rectum alone with its pathology, and encircle the anus with silver wire from below (Thiersch) or affix it to sacrum, abdominal wall, psoas muscle, broad ligament, etc., or cut it off without any thought or consideration of the pelvic floor.

It would seem that this finished product of nice adjustment after long centuries of preparation, has the rights of any other portion of the abdominal wall to claim, that when any organ in the abdomen has slipped its tether and passed through the cleft only intended for functioning passage, the dignity of recognition of a hernia and surgical restoration.

Prolapse of the rectum has not recognized over hundreds of years the necessity of the pelvic floor. The term "hernia of the rectum" recalls the work of vast centuries to prepare the pelvic floor to go with the rectum and look after its support and functioning. They belong together. The repair of one not only calls for the repair of the other, but the re-

attachment of the two is called for, which we must suggest has been overlooked in surgery of prolapse of the rectum.

and maldevelopment leave the pelvic floor weak. In a far greater number trauma and acquired weakness leave the pelvic
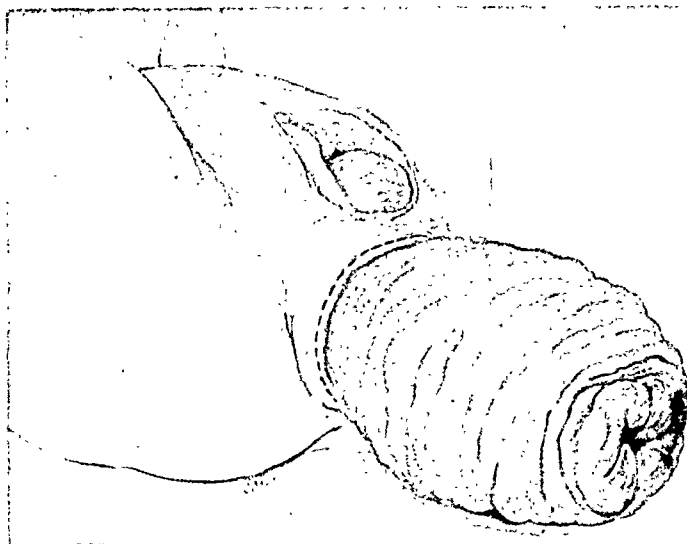
FIG. 1. Herniation of the rectum through the posterior cleft of the pelvic floor. A rectocele shows through the vulvar opening. This rectocele is increased in size when the herniation of rectum is reduced. Dotted lines showing incision at the mucocutaneous injunction.
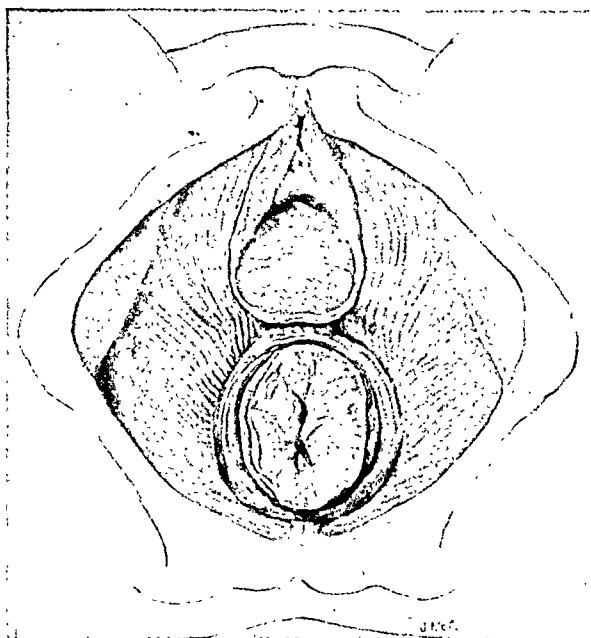
FIG. 2. Soft parts cut away showing greatly dilated sphincter and the levator muscles separated from pubis to coccyx from a case that would admit the hand or fist through the sphincter and pelvic floor.

The above arrangement for the support of the rectum provides for most cases, while in the minority, congenital defects

floor and its faulty attachment unable to withstand the combatting forces of frequent excessive strains, as in heavy lifting,

coughing, sneezing, straining with stone, stricture, enlarged prostate, constipation, diarrhea, etc., or pathological conditions medium size, that of an egg or orange size; or large size, that of a cocoanut or football or larger. The small or medium-sized

FIG. 3. A, the rectum being drawn down and sphincter being pushed back. As much rectum as thought necessary is removed. B, levator ani muscle being exposed through the sphincter muscle and being sewed anterior to the rectum. Mattress sutures being shown. C, rectum turned forward exposing the levator ani muscles posterior to the rectum, interrupted sutures here shown. Either suture may be used. In some extensive cases a line of mattress sutures and a line of interrupted sutures is used.

which begin primarily or secondarily in the bowel itself.

So the exigencies of the rectal and pelvic floor condition find us dealing with extrusions through the posterior pelvic floor slit with the rectal tissues becoming detached from the pelvic floor. This condition creates an increasing pathological disorder in both. The extrusion may be small, of a hickory or walnut size; of a ones may present fairly normal tissues and be ready for operation after a short period of preparation. The large ones are apt to be found in aged, weak individuals and exhibit extreme disorders, tardy to recover for safe operation. It is regrettable that any should reach the larger sizes of herniation. The smaller ones are much more amenable to ideal treatment, and the medium-sized hernia-

tions yield fairly well to preparation and operation. The treatment is based upon one's conception of the condition. If the
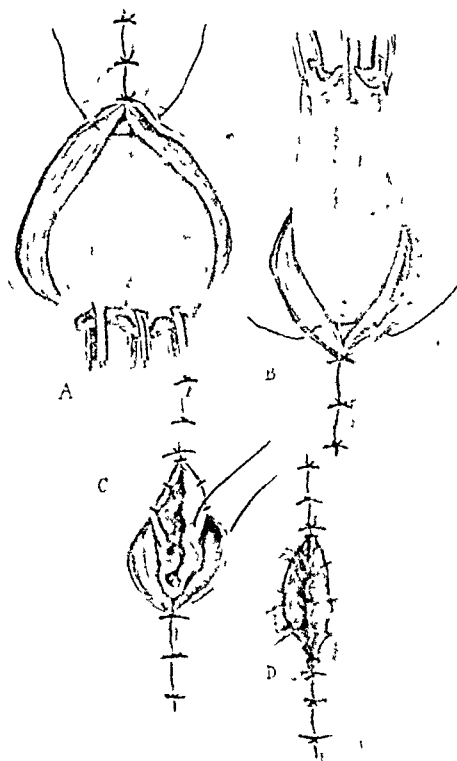
FIG. 4. A, shows rectum turned backward and sutures lessening the size of the skin and sphincter opening. B, the rectum turned forward showing the interrupted sutures lessening the size of the skin and sphincter openings posteriorly. C, shows rectum amputated and cut end being attached to the skin and sphincter on the sides. D, suturing of the skin and bowel completed leaving proper size sphincter and bowel opening.

treatment for prolapse of the rectum is, and always has been and will be directed at the bowel condition, the pelvic floor will be overlooked. If the condition is a herniation, the abdominal wall and the pathological status of the bowel will receive joint consideration.

In the determination of the correct surgical anatomy, we may well consider the words of Stone who says, "The term 'hernia' is used in general to designate the abnormal protrusion of organs or tissues through defects or openings that should enclose or confine them. . . . The factor of basic importance is not the sac or the contents, but it is the defect in the abdominal walls, floor or roof that allows protrusion of the abdominal contents." We have here then all the requirements of a hernia: a weakened wall and a gut pushing through. A hernioplastic repair of the pelvic floor, a bowel correction and an attachment of the bowel to the pelvic floor are necessary. This is accomplished as follows:

AN INTRA-SPHINCTERIAN HERNIOPLASTIC
OPERATION UPON THE PELVIC FLOOR
AND SPHINCTER, COMBINED WITH
AMPUTATION OF THE HERNIATED
PORTION OF THE BOWEL

1. Thorough preparation with as much preoperative improvement of the herniated structures as possible.

2. Anesthesia to suit the condition and the operator.

3. Lithotomy position, pelvis rather elevated and buttocks well over the edge of the table.

4. The herniation is preferably returned to pelvis, leaving the enlarged anal circle facing the operator like the face of a clock.

5. The cutaneo-anal junction is picked up with tooth forceps at the points 12, 3, 6 and 9 of the clock.

6. The line between 12 and 3 is put on the stretch and a sharp knife follows this line just inside the sphincter muscle; 3 to 6 is treated in the same way, then 6 to 9, then 9 to 12.

7. The cut end of the gut stands free in its whole circumference, when it is picked up by several tooth forceps and pulled toward the operator, while a knife severs any tough fibers and a sponge pushes back all structures surrounding the gut to the length that seems necessary to amputate the gut.

8. With the gut held intact, it is pulled backward as shown in the cut, and the sphincter is held forward while the enlarged pelvic floor ring is brought together

in front of the rectum with No. 2 interrupted catgut, the number of sutures varying from one to three, according to

united to the skin edges laterally and the operation is completed.

A smaller amount of gut will require
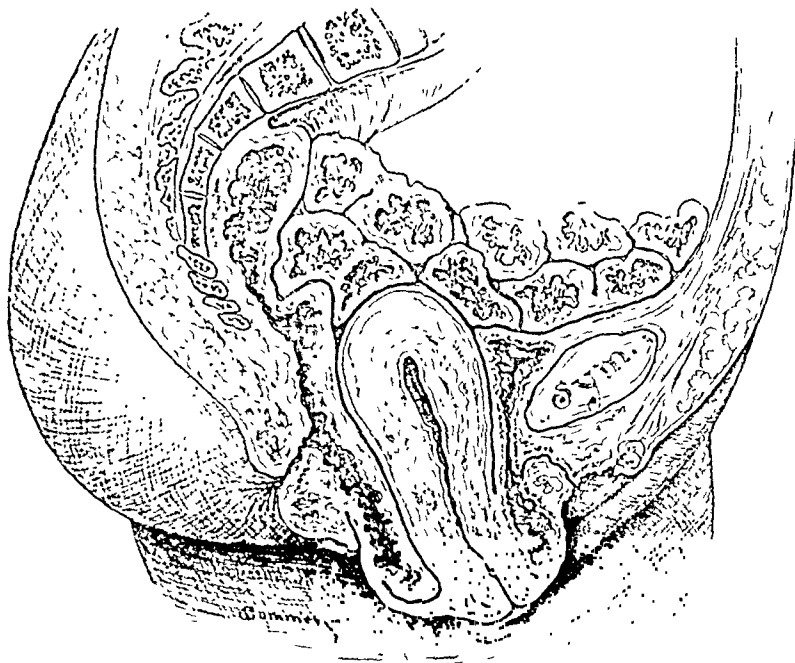
FIG. 5. Shows the rectal, bladder, uterine and peritoneal herniation through the anterior pelvic floor cleft. A little stretching of the sphincter in such a case would permit the rectum to herniate through the posterior cleft.

the size of the dilated ring. The posterior of these anterior sutures should take a grasp of the bowel at the level of the repaired pelvic floor.

9. The bowel is now turned forward over the repaired pelvic floor and the posterior portion of the pelvic floor ring is closed as described, anteriorly, preferably with two more sutures than in front. The most anterior suture of these posterior sutures grasps the posterior bowel wall, after which the side wall of the bowel is sewed to the remaining side of the cleft or hernial ring.

10. The considerably enlarged skin and sphincter circle is lessened, bringing them together in front of the rectum and posterior to the same, with two more sutures in front and two less posteriorly, to give the lower end of the bowel a slant backward, after which reduction in size. The proper amount of herniated gut is amputated, the free end of the gut is

amputation than is the case if no repair of sphincter and pelvic floor is done.

This now leaves only a healing by first intention to give a perfect functional and anatomical result. A moderate amount of suppuration may be compatible with a fair result.

The hernioplastic operation to cure rectocele is performed by raising the posterior vaginal flap and bringing the pelvic floor muscles and fascias together. Care should be taken to have the sutures reach far enough backward to get the muscles anterolateral to the rectum, in order to reduce the size of the cleft through which the rectum passes. Another precaution is to see that the anorectal structures are well attached to the supporting pelvic floor. In extensive bulging of the anterior rectal wall, transverse plication of the anterior rectal wall is recommended by Dannreuther, care being observed to avoid penetration of the bowel. It is of greatest

importance to use the muscles and fascias to reinforce such bulging rectal walls. Cases are seen in which a greatly stretched sittings could well be chosen if the nature of the work could easily find a dividing line.
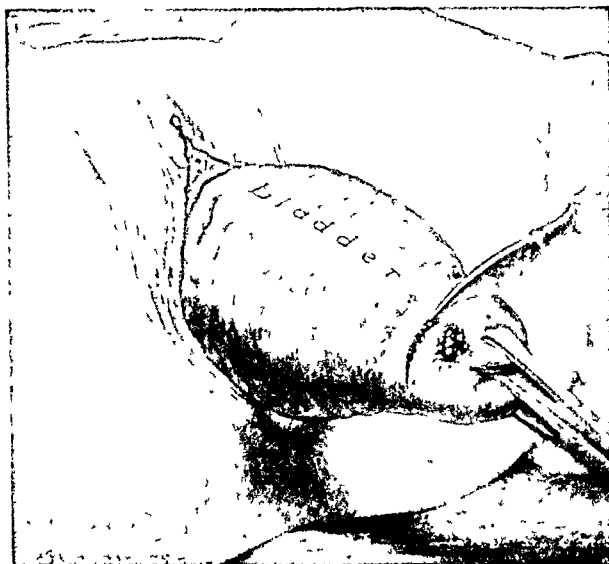
FIG. 6. Shows the same condition as is shown in cross section of Figure 5. Herniation of the uterus, bladder and rectum. With such loosening and coming down rectal herniation easily takes place.

rectovaginal septum may at one time herniate through the anterior cleft, and at another herniate through the posterior or anal cleft. Here the excess of vaginal flap may easily be cut away, but plication is more applicable for the rectal wall, provided rectal amputation is not demanded. In cases of complete tear with excess rectal wall, the anterior rectal flap may be drawn down and amputated. Some conservative operators consider rectal operating taboo after a line of vaginal repair, but not infrequently rectal repair is a part of vaginal repair.

Some pathologic conditions are not circumscribed by splitting the rectovaginal septum and some cases are found without a complete vaginal septum. I have in several instances amputated a moderately herniating rectum, and made a hernioplastic operation on the posterior cleft and sphincter, after a vaginal repair which included in some cases a vaginal hysterectomy. Cases and operators must be chosen for like extensive work and two

In cases not needing amputation, a strengthening of the pelvic floor and lessening of size of the sphincter circle is sometimes performed through a crescent-shaped incision, anterior or posterior to the anal opening. This procedure was elected in relaxed cases in Army work in France, and in one case of herniation with amputation, although the intrasphincterian hernioplastic repair would have been more suitable. Cuneo and Sénèque have more recently reported the method of anterior and posterior incision for reconstruction of sphincter control, but these alone are not applicable for herniation of considerable extent. Cunningham, Hartman and others have amputated the rectum without consideration of the muscle control. These cases could have the hernioplastic repair at the same time in a majority of cases, or at another time in extreme cases.

Cases could be appended to illustrate almost every phase of rectal herniation, but there are some cases that illustrate these herniations more than others. One

case that has come under my care so graphically demonstrates the hernial nature of the disorder and is so typical of development and means of relief, that it seems worthy of detail in place of a longer series.

A. G., age fifty-one, a doctor, formerly surgeon in the Russian Army, a well developed, athletic type, somewhat overweight, began to have herniation of the bowel when less than one year old. (Father had like condition as a child, but recovered.) Since then has never had a bowel movement without extrusion; extrusion and returns, two, three and four times before relief was obtained. Severe straining had developed double inguinal hernia. Each *hernia had returned in a short time after operation* had been performed by competent men. The patient, when a child, was taken to doctors by his father, but was advised to use temporizing means, hoping for improvement. Patient made a constant study of proposed measures of cure, but was not sufficiently impressed with the rationale. Over the years, and especially of late, the extruded mass became larger, and the sphincter perceptibly more dilated. More bleeding and more discharge was also noted.

The patient was impressed by the rationale of the intrasphincterian hernioplastic repair. After deciding upon operation, he took cleansing astringent enemas before entering the hospital and had four days' preparation in the hospital before operation. The bowel was kept inside by a large rectal catheter and antiseptic astringent solution was allowed to run in at frequent intervals. Sulfathiazole emulsion was used intrarectally and by external application. The rectal herniation was the size of a fist when the patient was brought to the operating room. Upon being reduced, the surface wrinkled and looked not unlike an overflowing cup of angleworms.

Operation was performed according to the method described. The wound closed, leaving a normal appearance, and healed by partial granulation. It was feared continence might not be perfect. The wound healing slowly, but in the end was well-nigh perfect. Sphincter control was good and the sphincter grasped the finger with force. Pelvic floor muscles, of the proper size were felt snugly grasping *the rectum. The rectum has not extruded* since the operation. Defecation and retention are normal, except slight leakage of liquid stools.

SUMMARY

1. We have undertaken to present these rectal extrusions as herniations through the pelvic floor.

2. The pelvic floor is the lower wall of the abdomen and as such is a support to the rectum when retaining its proper build and strength, and as a part of the wall is subject to weakness and hernial formation.

3. Hernioplastic repair of the pelvic floor and amputation of the extruded portion of the bowel is a rational procedure.

VESICO-INTESTINAL FISTULA—ACTUAL AND INCIPIENT

EARLY DIAGNOSIS AND TREATMENT

Joseph A. Lazarus, m.d. and Morris S. Marks, m.d.

NEW YORK, NEW YORK

THE subject of vesico-intestinal fistula has been frequently discussed in medical literature from a variety of angles. It shall be our purpose to treat this subject from the urological aspect, particularly with the view of emphasizing its early recognition in urologic diagnosis, and suggesting a few additional sidelights in treatment.

Fistulous communications between the bladder and the various segments of the intestinal tract are not uncommon, but still sufficiently infrequent to warrant comment. Higgins, for example, in 1936 was able to collect 583 cases, including thirty-five from the Cleveland Clinic. Kellogg, in 1938, reported 592 cases. Yet Balch, discussing Higgins' paper, stated that he was only able to find nineteen cases in going through the files of the record rooms of six Indianapolis hospitals having a total of 1,738 beds.

TYPES OF VESICO-INTESTINAL FISTULA

Higgins' grouping of cases is both simple and practical. He groups them as: (1) traumatic, (2) atraumatic or inflammatory, (3) tumors and (4) congenital. For our purpose we have found it somewhat more convenient to group the cases under two main heads, namely, (1) fistulas arising primarily from the bladder and (2) fistulas arising primarily from the intestine. Although this classification fails to take into account the group of traumatic cases as well as the small number of congenital cases, it is more pertinent to the text of this communication to limit our remarks to the groups of inflammatory and neoplastic lesions, since it is with these cases that the urologist is most apt to deal.

Fistulas may be actual or incipient. Actual or fully developed fistulas may occur in two forms: (a) direct communications between the bladder and bowel through short straight sinuses, and (b) long narrow tortuous sinuses connecting both viscera. The former is usually seen in cases due to carcinoma and tuberculosis, while the latter occurs most frequently in cases resulting from infections. Males are more frequently affected than females in the proportion of 3 to 1 (Pascal). The reason for this discrepancy has been attributed to the intervention of the uterus between the bladder and the intestine, and to the prevalence of diverticulitis of the sigmoid in the male. The greatest incidence is between the ages of fifty and sixty.

A careful analysis of the cases collected by Higgins, Kellogg and Balch seems to indicate that inflammatory lesions of the bowel constitute the underlying cause of the fistulas in the majority of cases (51 per cent), while tumors of the bowel were the underlying cause in 21.3 per cent. The bladder was the primary site of the lesion in 11.6 per cent of cases.

Higgins' statistics seem to indicate that vesical calculi constituted the most frequent cause of fistula formation in the bladder group (39.3 per cent), while carcinoma ranked second with 30 per cent. Vesical diverticula were found in 24.2 per cent of the cases.

An analysis of the inflammatory intestinal group showed diverticulitis to have been the primary cause in 65.8 per cent, tuberculosis was found in 17.8 per cent and appendicitis was present fourteen times (9.5 per cent) (Higgins). In Kellogg's series appendicitis was present in only 4 per cent of cases, while the incidence of

appendicitis in Balch's group was 25 per cent.

In Higgins' series the most frequent neoplastic lesion of the bowel was carcinoma of the rectum (50.8 per cent), as compared with 11.8 per cent in Balch's group. Carcinoma of the sigmoid was present in 45.9 per cent of the cases in Higgins' collection.

As regards the location of the fistula, the rectum and bladder were involved in 43.5 per cent (Higgins); 47.4 per cent (Balch); and in only 16 per cent (Kellogg). A fistulous communication between bladder and sigmoid was present in 23.8 per cent (Higgins), 26.3 per cent (Balch) and 63 per cent (Kellogg). The incidence of ileo-vesical fistula was 4.1 per cent (Higgins), 15.7 per cent (Balch) and 4 per cent (Kellogg). A communication between the bladder and the appendix was found fifteen times (3.4 per cent) in Higgins' series; twice (10.6 per cent) by Balch and in 4 per cent of the cases collected by Kellogg. Higgins noted eighty-two instances of vesicocolonic fistulas in a total of 441 collected cases (18.6 per cent).

SYMPTOMS

Actual Fistulas. The passage of gas or stool through the urethra constitutes unequivocal evidence of a vesico-intestinal fistula. Although the passage of urine from the rectum is also pathognomonic of this condition, this finding is not as readily ascertained as the former. It is, of course, obvious that collateral symptoms are present which in turn depend upon the segment of bowel with which the bladder communicates. If the initial lesion happens to be a carcinoma of the rectum which has subsequently broken into the bladder, the presenting symptoms will be those characteristic of rectal carcinoma, namely, constipation alternating with diarrhea, blood and mucus in stools and loss of weight. The same holds true when the initial lesion is diverticulitis of the sigmoid, in which event the outstanding symptoms are pain in the left lower quadrant of the

abdomen, with blood, pus and mucus in the stool. Symptoms referable to the bladder depend upon the degree of involvement present. With the actual passage of intestinal contents into the vesical lumen, acute cystitis invariably ensues, with urinary frequency, dysuria and urgency as outstanding symptoms. The urine contains blood, pus and intestinal contents. While the passage of gas in the urine is extremely suggestive of a vesico-intestinal communication, it is occasionally found in diabetic patients with infected urines. Should an ascending infection occur, the picture is that of an acute pyelonephritis with chills, fever and lumbar pain. When the lesion is primarily in the bladder, vesical symptoms naturally predominate, and in fact will most frequently completely mask the intestinal symptoms. Diarrhea with or without tenesmus is not an uncommon symptom, particularly when an ample flow of urine into the bowel is present.

Incipient (Threatened) Fistulas. In the event the bladder is the primary seat of the lesion, it is extremely difficult to outline a syndrome which is at all suggestive of a threatened communication with an adjacent segment of bowel, since the bladder lesion must be extensive before it can lead to a vesico-intestinal fistula. Well defined urological symptoms are present long before threatening perforation occurs, so that from the urological standpoint, one can hardly suspect a threatened break-through to be detectable by changes in the genitourinary symptoms. Suspicion might be directed to this possibility, however, by the sudden onset of intestinal symptoms in a patient suffering from a serious vesical lesion.

When the lesion originates in a segment of bowel, an incipient or threatened breakthrough can and should be recognized promptly, since by so doing serious consequences may be averted. Given a patient with carcinoma of the rectum, or diverticulitis of the sigmoid, or terminal ileitis, who suddenly develops acute urological symp-

toms, it is encumbent upon the surgeon to seek an explanation for such symptoms. Symptoms may be extremely mild, such as slight increased frequency of urination or mild burning on urination; or the only suggestion of a urinary abnormality may be a varying degree of pyuria which may be constant or intermittent.

DIAGNOSIS

Actual Fistulas. In cases of well defined fistulas, the diagnosis can readily be established when patients pass urine in the stool, or gas and fecal matter through the urethra. This, unfortunately, is not always the case, in which event it becomes necessary to resort to other diagnostic procedures.

Cystoscopy usually reveals a marked degree of cystitis. The site of the communication appears as an area of edema which surrounds the actual point of perforation. The latter may or may not be seen. The stool may be seen passing through the actual fistula, or lying free in the bladder.

Occasionally, one may clinch the diagnosis by filling the bladder with a solution of indigo-carmine, and watching it return through a rectal tube or proctoscope previously introduced into the rectum.

Although theoretically possible, it is extremely difficult to visualize the rectal or sigmoidal opening of the fistula through the sigmoidoscope. Instances have been reported in which the diagnosis was established by means of the barium enema.

Incipient Fistulas. The diagnosis of a threatened or incipient fistula can only be made by proper cystoscopic examinations. The earliest sign discernible by cystoscopy is a small localized area of redness somewhere on the posterior wall of the bladder, in an otherwise normal appearing viscus. At a slightly later stage, this area reveals itself as an island of bullous edema which, when the bladder is filled to capacity, may be completely or partially ironed out. Shortly before perforation occurs the lesion closely simulates a bladder tumor because of the presence of papillomatous-like ex-

crescences in the region of the bullous edema. The vesical mucosa surrounding this lesion may, at this time, reveal a moderate degree of erythema. An important diagnostic feature at this stage is a history of intermittent clouding of the urine. Should the urine during a cystoscopy be found turbid due to pus, the ureters should invariably be catheterized. Negative urines obtained from both kidneys, microscopically and culturally, serve as confirmative evidence that the source of the pyuria is the bladder, and lends additional weight to the suspicion that the bladder lesion is due to an incipient communication with a segment of bowel.

Localizing the exact site of the fistulous opening in the bladder is of little diagnostic importance because of the extreme mobility of the small bowel, sigmoid, transverse colon and appendix, and also because of the fact that in well established cases the high grade of accompanying cystitis and reduced vesical capacity make localization difficult and inaccurate.

PROGNOSIS

Prognosis in these cases depends upon a variety of factors. The character of the primary lesion responsible for the fistula is extremely important in appraising the ultimate prognosis. If the lesion is a malignant one, the outlook is naturally poor. On the other hand, should the original lesion be inflammatory, such as an appendiceal abscess, the outlook is good. The presence or absence of genitourinary complications such as unilateral or bilateral pyelonephritis also materially influences the prognosis, and this in turn is dependent upon the duration of the disease. In cases of threatened or incipient fistulas, the prognosis, particularly the immediate prognosis, is far better than in cases of well established vesico-intestinal fistulas. Another important consideration is the age and physical status of the patient, as younger people stand surgical intervention better than older ones. The presence of cachexia is not conducive to good healing.

TREATMENT

Vesico-intestinal fistula is an outright surgical disease and treatment may be palliative or curative. Palliative treatment is indicated in hopeless cases of carcinoma of the bowel or bladder, in which event colostomy or cecostomy is the procedure of choice in diverting the fecal flow from the bladder. Palliative colostomy may also be tried in certain cases of vesico-intestinal fistula resulting from diverticulitis of the colon. In these cases the colostomy, once established, must not be closed for many months, in order to give the involved bowel full opportunity to heal spontaneously. Colostomy is also indicated in hopeless cases of carcinoma of the bladder associated with vesico-intestinal fistula.

Curative treatment will, of necessity, depend upon the underlying pathological condition. In cases of threatened perforation, it is our opinion that preliminary colostomy is not necessary, but that one can, after proper preparation of the patient, perform laparotomy and direct appropriate treatment to the underlying lesion. By proper preparation of the patient, we mean at least four to six days of treatment with the patient in a hospital. He is given a high caloric, low residue diet containing high vitamins and minerals, and thorough cleansing of the bowel by daily use of saline catharsis. At least 2,000 cc. of 5 per cent glucose in normal saline is administered daily by slow intravenous drip, and 4 Gm. of sulfaguanidine given by mouth every eight hours, or sulfadiazine in 15 gr. doses at four hour intervals. At least one transfusion of 500 cc. of citrated blood is given before operation and another transfusion immediately after operation. In cases of carcinoma or diverticulitis of the sigmoid, the affected segment of bowel is carefully dissected away from the bladder wall to which it is more or less firmly adherent, by blunt finger dissection, and whenever possible a multiplestage Mikulicz procedure is carried out. When the lesion is due to carcinoma of the rectum one may,

after carefully dissecting the bowel away from the bladder, carry out a Miles abdominoperineal resection in one or two stages, depending upon the patient's physical condition. The Mummary operation is also an excellent procedure in poor risks. When the attachment between the bladder and rectum is due to inflammatory adhesions, the bladder need not be disturbed. In cases in which the vesical wall is invaded by carcinoma, a segment of bladder can be removed along with the tumor and the bladder reconstructed over a large suprapubic drainage tube.

In cases of threatened vesico-intestinal fistulas caused by appendiceal abscesses, the abscesses should be drained and the appendiceal stumps removed whenever possible. In cases of terminal ileitis, immediate transverse ileocolostomy is carried out, after completely transecting the ileum at a sufficient distance from the inflamed segment of bowel in order to make certain that the anastomosis is performed upon healthy intestine. If it is deemed safe to complete the operation in one stage, the segment of ileum attached to the bladder is carefully dissected away from the bladder and removed along with the cecum and ascending colon. Should a two-stage procedure be considered advisable, the segment of ileum attached to the bladder is left undisturbed, and removed at the time of the second stage of the operation.

In the treatment of actual or true vesico-intestinal fistulas, it is our belief that a preliminary colostomy is necessary: (a) when actual stool is present in the urine; (b) when pyelonephritis is present; (c) when the basic lesion is carcinoma, either of the bladder or of the bowel; (d) when the bladder is markedly inflamed and contracted; (e) in cases of marked debility; and finally (f) in cases in which laparotomy discloses a lesion of the bowel, the identity of which is not readily discernible, or in which the bowel is so intimately adherent to the bladder that detachment is considered too hazardous. In performing such a colostomy for diversion of the fecal

stream, it is best to avoid a loop colostomy. A double barrel colostomy such as advocated by Lahey or Devine seems best suited for this purpose. When the lesion is hopelessly inoperable the colostomy becomes a permanent one.

As regards the time interval that should be permitted to elapse before attacking the lesion, it is our opinion that it is best to err on the long side rather than to operate too soon. This is particularly true when dealing with inflammatory lesions. In cases of carcinoma one can safely wait six to eight weeks.

A pertinent problem in this type of case is the method of handling the opening in the bladder wall. We believe that when the opening is small it can safely be left undisturbed, and an indwelling catheter introduced into the bladder through the urethra and left *in situ* for a week or ten days. In cases of larger openings, we have found that as a rule sutures do not hold because of the friability of the bladder wall, and we, therefore, resort to suprapubic cystotomy with adequate drainage of the cul-de-sac of Douglas to provide for any possible leakage.

We have found the local use of crystalline sulfanilamide of inestimable value in handling such fistulas. We do not hesitate using 12 Gm. of the drug in the cul-de-sac after detaching the bowel from the bladder wall. Following operation patients are routinely given 15 gr. of sulfadiazine at four hour intervals for at least four to five days.

Since, in most instances, the vesical capacity is markedly reduced in cases of actual vesico-intestinal fistulas, we consider it advantageous to dilate the bladder with hydraulic pressure within five to six weeks after the wound has healed. This procedure must be cautiously carried out to avoid throwing too much strain upon the weakened bladder wall.

CASE REPORTS

CASE I. Mr. S. F., aged sixty-one, first seen January 10, 1939, complained of prostatism and bilateral lumbar pain of two years' dura-

tion. Fifteen years previously he passed several calculi. There were no gastrointestinal symptoms. Following a complete study of the genitourinary tract, the diagnosis made was benign hypertrophy of the prostate. A two-stage suprapubic prostatectomy was performed and he was discharged from the hospital after twenty-one days.

About twenty-one months later transurethral revision of the bladder neck was performed for a small recurrent adenoma of the right lobe of the prostate. He left the hospital symptom-free seven days later. On January 31, 1942, approximately three months after the revision, he returned complaining of increased urinary frequency and pyuria. At that time cystoscopy disclosed an edematous circular lesion with a central umbilication situated on the posterior surface of the bladder just above the inter-ureteric ridge. Our impression at the time of this examination was that we were dealing with an extravesical lesion which was threatening to penetrate into the lumen of the bladder. A careful digital rectal examination was negative. Sigmoidoscopy failed to show the presence of a tumor, but revealed a slight reddening and edema of the rectal mucosa. Barium enema x-ray and fluoroscopy showed the sigmoid flexure drawn over toward the cecum, but failed to reveal a defect. His weight was 177 pounds.

When seen ten days later he stated that although he noticed no urinary discomfort, the urine appeared very turbid at times, and at other times it was comparatively clear. Cystoscopy disclosed the same polypoid lesion seen at the previous cystoscopy. Although resembling a bladder tumor, the lesion was considered entirely inflammatory. Overdistending the bladder failed to completely iron out the lesion. The urine contained much pus. Two weeks later hydraulic distention of the bladder through the cystoscope showed that the lesion could be ironed out when the bladder was fully distended, leaving behind a slight nodular thickening of the mucosa at the site of the lesion. Both kidneys were catheterized and the specimens were found negative microscopically and culturally. At this time he was carefully questioned as to whether he had passed any feces or gas in the urine, but he did not recall such an occurrence. One week later he returned complaining of vesical irritability. A punch biopsy was performed and the tissue

report was "edematous, chronically inflamed bladder wall." Rectal examination was entirely negative, and there were no symptoms referable to the gastrointestinal tract. It appeared to us then that we were dealing with a threatened vesico-intestinal fistula and laparotomy was advised and accepted. He entered the hospital March 29, 1942, for preoperative preparation which consisted of thorough cleansing of the bowel, low residue, high vitamin diet and sulfaguanidine 4 Gm. every eight hours. The day prior to operation he was given 500 cc. of citrated blood.

Operation. (J. A. L.) On April 2, 1942, under spinal anesthesia the abdomen was opened through a long left paramedian suprapubic incision extending from the symphysis to a point just above the level of the umbilicus. There was a large infiltrating mass about the size of an adult's fist situated deep in the pelvis, intimately adherent to the posterior wall of the bladder. The mass consisted of the lower sigmoid flexure directly above the rectosigmoidal juncture. The liver and para-aortic lymph-nodes were not involved.

With the patient in high Trendelenburg position the intestines were carefully walled off with hot pads, and by careful digital manipulation the sigmoid was easily dissected away from the posterior wall of the bladder. Although the vesical wall was indurated, no opening could be visualized. There was no evidence of free pus during this procedure. Owing to the low position of the tumor it was necessary to divide the peritoneal reflexion on both sides of the mesentery as well as the peritoneal reflexion over the bladder in order to obtain sufficient mobility of the involved segment to perform a Mickulicz operation. The loop bearing the tumor was then easily delivered from the abdomen, and after uniting both limbs and closing the layers of the abdominal wall snugly around the exteriorated loop, the tumor was removed between clamps. The proximal and distal clamps were left *in situ,* thereby completely obstructing the bowel. Three Gm. of sulfanilamide powder were placed into the pelvis and around the wound. No attempt was made to reinforce the portion of the bladder wall freed from the tumor, but an indwelling urethral catheter was introduced and left *in situ* for seven days. The patient is making an uneventful convalescence and is awaiting the crushing of the spur.

Pathological diagnosis of the extirpated segment of bowel was "infiltrating adenocarcinoma of rectum with extension into adjacent fat tissue."

Comment. This is an example of a case of threatened vesico-intestinal fistula caused by carcinoma of the rectosigmoid, in which the cardinal symptoms were entirely vesical, namely, intermittent pyuria with attacks of vesical irritability. Cystoscopically, there was a polypoid edematous lesion on the posterior vesical wall simulating a bladder tumor, which at first could be almost completely ironed out by hydraulic distention of the bladder, but later could not be obliterated. Although no distinct communication between the bowel and bladder was found, it was difficult to explain the intermittent attacks of pyuria without assuming the presence of some, possibly minute, sinus between the two viscera, not large enough to permit the passage of gas or stool, but capable of transmitting bacteria. A tentative diagnosis of a threatened vesico-intestinal fistula was made prior to surgery. This enabled us to institute proper preoperative preparations. It is our opinion that such preparation was greatly responsible for the patient's smooth postoperative convalescence.

CASE II. R. C., male, aged twenty-four, first seen December 16, 1940, complained of hematuria and right renal colic, dysuria and increased urinary frequency of two weeks' duration. His past history was essentially unimportant, save for an appendectomy performed in 1937.

Cystoscopy revealed a lesion the size of a fifty-cent piece occupying the right posterior wall of the bladder, which had the appearance of an irregular tumefaction with a central umbilication, attached to the bladder by a broad base. There was no inflammatory reaction of the vesical mucosa surrounding the base of the lesion. The question that arose was whether we were dealing with an infiltrating bladder tumor or with a threatened vesico-intestinal fistula. Both kidneys were easily catheterized, and the specimens from both sides showed an occasional leucocyte. Urinal-

ysis: specific gravity 1.025; albumin 1 plus; sugar negative; micro-mass of white blood cells. The bladder specimen showed Gram-negative bacilli on smear and Bacillus coli aerogenes on culture. The renal specimens were negative on smear and sterile on culture. All specimens were negative for the mycobacterium tuberculosis.

Blood Chemistry: urea nitrogen 11.5 mg. per cent; creatinin 1.07 mg. per cent; urea clearance 127.5 per cent. The genitourinary tract failed to show any abnormality on the flat and pyelographic x-rays.

The following day biopsy was performed upon the vesical lesion. This was reported inflammatory with no evidence of malignancy.

Physical examination was essentially negative, except for a large, boggy prostate which was felt per rectum.

Preoperative Diagnosis: Threatened vesico-intestinal fistula. The patient was sent to the hospital and thoroughly prepared for 6 days prior to operation.

Operation. (J. A. L.) On January 9, 1941, under spinal anesthesia the abdomen was opened through a six-inch midsuprapubic incision. Starting at approximately six inches from the ileocecal valve, the terminal ileum was intimately adherent to the posterior wall of the bladder. At this point the ileum was angulated and the proximal limb of this loop, which was adherent to the bladder, was dilated. The wall of this portion of ileum was very friable. The proximal limb of the loop was mobilized at a distance of about twelve inches from its attachment to the bladder and divided between crushing clamps, along with its mesentery. Both ends of the severed intestine were inverted with three layers of sutures. The proximal segment was then anastamosed side-to-side with the midportion of the transverse colon. The wound was closed in layers.

On January 27, 1941, eighteen days after the ileocolostomy, he was given 500 cc. of citrated blood and the following day, under spinal anesthesia the abdomen was opened through a long right rectus incision extending from the level of the costal arch to the symphysis. There were dense adhesions binding the terminal ileum to the posterior bladder wall. After carefully separating this loop of intestine from the bladder by blunt dissection, a small perforation was seen in the center of an area of indurated vesical wall. The affected terminal segment of ileum, ascending and proximal part of the transverse colon were resected *en masse*, and the end of the colon inverted with three layers of sutures. Raw surfaces were obliterated by suturing the cut edges of the posterior parietal peritoneum. A small Penrose drain was introduced into the retroperitoneal space and permitted to emerge from the upper angle of the wound. An attempt to close the fistulous opening into the bladder was unsuccessful due to induration and friability of the tissue. A rubber dam was introduced into the pelvis behind the bladder and permitted to emerge from the lower angle of the wound. The wound was closed in layers around drains. An indwelling urethral catheter was introduced and left *in situ* for seven days.

Following a completely uneventful recovery, he was discharged from the hospital February 12, 1941. When last seen September 18, 1941, he was completely symptom-free and had gained fifteen pounds in weight.

Comment. This is a case of threatened vesico-intestinal fistula due to terminal ileitis, in which the diagnosis was made prior to operation by cystoscopic examination. The symptoms were entirely urological. Complete recovery followed a two-stage resection and transverse ileocolostomy. The opening in the bladder could not be closed by suture, but healed spontaneously with the aid of an indwelling urethral catheter.

CASE III. G. W., male, aged forty, first seen November 1, 1932, complained of severe lower abdominal pain, with complete obstipation of two days' duration. Urinary symptoms consisted of slight dysuria and increased urinary frequency. Preoperative diagnosis: Intestinal obstruction.

Laparotomy performed on November 1, 1932, revealed a large carcinoma involving the sigmoid, which was intimately adherent to the posterior vesical wall. The terminal four inches of ileum were also glued to the bladder wall. A transverse colostomy was performed. Death followed one week later, apparently from severe hemorrhage in the abdominal wound.

Autopsy showed no abnormality of the stomach and small intestine. A colostomy opening was present in the transverse colon.

The lower bowel presented many adhesions and the sigmoid was densely adherent to the bladder. There was a ragged, necrotic, annular, infiltrating tumor in the sigmoid, which had invaded the pelvic fascia and had perforated into the vesical lumen.

Microscopic examination of the sigmoidal lesion showed a papillary tumor forming irregular acini. There was great variation in the size and shape of the cells. The stroma was moderate in amount. Microscopic examination disclosed the bladder wall thickened and diffusely infiltrated with tumor cells of sigmoidal origin. The vesical mucosa in the region of the fistula was markedly hyperplastic.

Comment. This patient presented himself with symptoms of acute intestinal obstruction due to carcinoma of the sigmoid. The urological symptoms were mild, consisting of slight dysuria and increased urinary frequency. There was no evidence of stool or of the passage of gas through the urethra. Yet at operation it was noted that the tumor of the sigmoid was intimately adherent to the bladder. At autopsy an actual fistula between the sigmoid and bladder was found.

Case iv. J. L., male, aged forty-three, a resident of Honduras, Central America, first seen July 10, 1940, complained of passing stool and gas through the urethra. Illness began six years previously with difficulty in voiding and the passage of stool in the urine. Since then he had had intermittent bouts of chills and fever. He voided frequently day and night, had dysuria, and passed ribbon-like stools. His appetite was poor and bowels costive. There was a loss of fifteen pounds in weight during the past six months. The patient admitted that he had contracted syphilis twenty-five years previously and gonorrhea eighteen years ago.

Cystoscopy. A No. 21 F. cystoscope was passed with difficulty due to an obstruction at the vesical neck. The bladder capacity was very much reduced and there was a marked degree of cystitis. There was a large tumefaction involving the posterior wall of the bladder, extending into the vesical lumen. A well defined median bar was also present. Biopsy of the posterior vesical wall was reported

"bladder epithelium showing extensive acute and chronic inflammation."

X-ray of the Genitourinary Tract. The right renal silhouette was much enlarged while the left appeared normal. Intravenous pyelography disclosed delayed filling of a large right hydronephrotic kidney. The left renal pelvis showed no abnormality. Cystogram showed a filling defect involving the vault and right half of the bladder.

Barium enema showed a small amount of barium filling the ampulla and rectosigmoid. The barium appeared to pass over to the right side of the pelvis, where it seemed to fray out and blend into the adjacent tissue.

Sigmoidoscopy disclosed a markedly thickened and edematous rectal mucosa but no evidence of tumor.

Physical examination was essentially negative.

Laboratory Findings. Urinalysis: specific gravity 1.012; albumin 2 plus; sugar negative; microscopy—pus and bacteria. Blood chemistry: urea n. 12.5 mg. per cent; creatinin 1.0 mg. per cent; urea clearance 136 per cent. Bacteriology: bladder urine showed Staphylococcus albus on culture. Blood count: hemoglobin 11.1 Gm. per 100 cc. of blood; red blood cells 3,225,000; white blood cells 8,250 per cu. mm.; color index 1.2. Differential: polymorphonuclear cells 66 per cent; lymphocytes 11 per cent; rods 19 per cent; mononuclears 4 per cent. Wassermann was 2 plus. 2 hour phenolsulphonphthalein 30 per cent.

Preoperative Diagnosis. Actual vesico-intestinal fistula, possibly due to carcinoma of sigmoid, with right infected hydronephrosis.

Operation I. (J. A. L.) On July 20, 1940, under spinal anesthesia the abdomen was opened through a six-inch left reversed Kammerer incision. Situated in the pelvis was a large mass consisting of rectosigmoid intimately adherent to the bladder. Although a specimen for biopsy could not be obtained, it was believed that the lesion was inflammatory rather then neoplastic. A tranverse colostomy was performed.

Operation II. (J. A. L.) On August 6, 1940, owing to the patient's marked intolerance to an indwelling urethral catheter and to the severe grade of cystitis present, a suprapubic cystotomy was performed. Upon opening the bladder a small polypoid lesion was noted on the posterior wall of the bladder. In the center

of this lesion there was a small opening through which a ribbon of pus could be seen escaping into the bladder cavity.

Operation III. (J. A. L.) On August 23, 1940, under spinal anesthesia a right nephrectomy and subtotal ureterectomy was performed for infected hydronephrosis.

He was discharged from the hospital on September 23, 1940, and returned to Honduras with a well regulated functioning colostomy. He returned to New York City on April 25, 1941, and was admitted to the hospital in preparation for a segmental resection of the sigmoid.

Operation IV. (J. A. L.) On May 6, 1941, with the patient in high Trendelenburg position, the abdomen was opened through a ten-inch right reverse Kammerer incision extending from the symphysis to a point two inches above the umbilicus. The sigmoid flexure was found indurated and adherent to the posterior wall of the bladder. By gentle finger dissection the sigmoid was carefully peeled away from the bladder. A segmental resection of the involved portion of the sigmoid was then performed, and an end-to-end anastomosis accomplished. No effort was made to close the defect in the bladder wall because of the indurated state of the tissues around the fistula. An indwelling urethral catheter was introduced. Sulfanilamide crystals were placed into the pelvis and appropriate drainage instituted. Following the operation he was given 500 cc. of citrated blood. Following an uneventful convalescence he was prepared for closure of the colostomy which was performed on June 13, 1941. He was discharged from the hospital symptom-free and all wounds healed on July 5, 1941.

Comment. We have here discussed a case of actual vesico-intestinal fistula resulting from diverticulitis of the sigmoid, complicated by a severe infection of one kidney. The stages employed in handling such a problem have been carefully reviewed. No attempt was made to close the vesical opening, which healed spontaneously with the aid of an indwelling urethral catheter. It is our impression that the use of sulfonamides both orally and locally played an extremely important rôle in the smoothness of the postoperative

convalescence following the segmental resection of the involved sigmoidal segment of colon.

CASE V. S. R., male, aged sixty-four, was admitted to the hospital on the night of April 28, 1941, with an acute attack of urinary retention of twenty-four hours' duration. During the past few months he had been complaining of increased diurnal and nocturnal urinary frequency. On careful questioning he admitted a loss of twenty-five pounds in weight during the past year. Two weeks previously he began having nausea and vomited on a few occasions, and also complained of alternating bouts of diarrhea and constipation.

Physical examination disclosed an emaciated, cachectic looking individual who appeared in great distress. Rectal examination failed to disclose a lesion within the lower bowel. The prostate felt neither hard nor enlarged, but a boggy fullness could be made out in the cul-de-sac. On abdominal palpation an indefinite mass was felt in the region of the left iliac fossa. The rest of the examination was essentially unimportant.

Cystoscopy revealed a well defined median bar and moderate contracture of the vesical neck. There was no evidence of lateral lobe enlargement. Situated on the posterior wall of the bladder near the vault was an area of redness and edema suggesting the possibility that this portion of the bladder was in contact with some affected intra-abdominal viscus.

Sigmoidoscopy. Situated four and a half inches from the anus and involving the anterior wall of the rectum there was an ulcerating carcinoma which, on biopsy, proved to be adenocarcinoma.

Operation. (J. A. L.) On May 13, 1941, under spinal anesthesia the abdomen was opened through a long left rectus incision, extending from the symphysis to the level of the umbilicus. Situated in the pelvis there was a large indurated mass about the size of an orange, consisting of rectosigmoid hopelessly adherent to the posterior wall of the bladder, and a loop of ileum. The entire mass was also adherent to the anterior parietal peritoneum. A permanent colostomy was performed, using the sigmoidal loop.

Following the operation all urinary symptoms promptly disappeared, and the patient lived in relative comfort for seven months before he succumbed.

Comment. This illustrates another case of incipient vesico-intestinal fistula due to carcinoma of the rectosigmoid, giving rise to symptoms of prostatism and complete urinary retention. Owing to the marked discrepancy between the urinary symptoms and the cystoscopic findings, and also because of the presence of a characteristic area of edema and congestion on the posterior vesical wall, a tentative diagnosis of an intra-abdominal lesion secondarily affecting the bladder was made. This was confirmed at operation. Complete diversion of the fecal flow from the affected segment of bowel by permanent colostomy led to prompt abatement of urinary symptoms, indicating thereby the fact that irritation of a portion of the detrusor muscle by an extravesical lesion can and does give rise to symptoms of prostatism indistinguishable from a similar syndrome caused by a lesion of the prostate gland. The differential diagnosis can only be made by careful cystoscopic interpretation.

SUMMARY AND CONCLUSIONS

1. Vesico-intestinal fistulas may be divided into actual fistulas and incipient or threatened fistulas.

2. Careful analysis of the literature indicates that inflammatory lesions of the bowel constitute the underlying cause of fistulas in the majority of cases (51 per cent). Tumors of the bowel were present in 21.3 per cent of the collected cases. A review of the inflammatory intestinal group shows that diverticulitis was the primary cause in 65.8 per cent of the cases. The most frequent neoplastic lesion of the bowel was carcinoma of the rectum (50.8 per cent). As regards the location of the fistula, it was noted that the most frequent site was between the bladder and rectum (43.5 per cent).

3. The cardinal symptoms of actual fistula are the passage of gas or stool through the urethra. Symptoms of an incipient or threatened fistula are more suggestive than pathognomonic. In cases in which the primary lesion is in the bladder, the appearance of intestinal symptoms is suggestive of a contact inflammation between the bladder and intestine. When the primary lesion is in the bowel, the appearance of vesical symptoms should likewise suggest this same condition.

4. Diagnosis, especially in cases of incipient fistulas, depends primarily upon cystoscopic examination.

5. Proper preoperative preparation of the bowel is probably one of the most important requirements for successful surgery in this type of case. In cases of actual fistulas, graded stage operations are necessary. The use of an indwelling urethral catheter obviates the necessity of closing the fistulous opening in the bladder. The systemic and local employment of sulfonamides has been found of great value in this group of cases.

6. Prognosis depends entirely upon the underlying pathological condition.

REFERENCES

BALCH, J. F. *J. Urol.,* 86: 706–707, 1931.
HIGGINS, C. C. *J. Urol.,* 86: 694–705, 1931.
KELLOGG, W. A. *Am. J. Surg.,* 41: 135–186, 1938.
PASCAL, A. Thèse de Paris, 1900.

ABSCESSES ABOUT THE ANORECTUM

STUART T. ROSS, M.D.

Instructor, Department of Proctology, New York Polyclinic Medical School and Hospital; Assistant Attending Surgeon in Proctology, Methodist Hospital, Brooklyn

HEMPSTEAD, NEW YORK

ABSCESSES about the anorectum are among the more frequent conditions seen in medical practice. It is surprising, therefore, that there should exist such a confusion concerning the diagnosis, prognosis and proper treatment of these lesions.

ANATOMY AND CLASSIFICATION

The key to the understanding and successful treatment of anorectal abscesses lies in a knowledge of the anatomy. Infections occur in well recognized locations and mainly in pre-existing potential spaces. For this reason, a brief review of the essential anatomy is in order.

The levator ani muscle is the dividing line between the deep and superficial structures of the region; it arises from the lateral pelvic wall, coursing medially and inferiorly to insert into the midportion of the anal canal between the internal and external sphincters. (Fig. 1.) Below this muscle, on each side of the anus, is an ischiorectal space, bounded by the obturator internus laterally, the skin and subcutaneous tissues inferiorly and the fascia lining the undersurface of the levator superiorly and medially. The ischiorectal space is, then, roughly pyramidal in shape, and filled with an elastic pad of fat.

Above the levator, there is a potential space between the superior fascial covering of the muscle and the lowermost reflection of the peritoneum. This space is limited posterolaterally on each side by the lateral ligaments of the rectum, in which course the middle hemorrhoidal vessels, and is called the pelvirectal space. Between the two lateral ligaments posteriorly, that is, between the rectum anteriorly and the sacrum posteriorly, is the postrectal or retrorectal space.

Abscesses may be classified according to their location as follows:

1. *Deep* (above the levator)
   a. Submucous (in the rectal wall)
   b. Pelvirectal (in the superior pelvirectal space)
   c. Retrorectal (in the postrectal space)
2. *Superficial* (below the levator)
   a. Intracutaneous
   b. Subcutaneous
   c. Ischiorectal

ETIOLOGY AND PATHOGENESIS

The sequence of events in nearly all of the more common abscesses, is quite uniform. At the anorectal line, which is located 1½ to 2 inches from the anal verge, is a series of small pockets, circularly disposed, and called crypts of Morgagni. These pockets are normally only 1 to 2 mm. in depth. They open upward toward the rectum, and are equipped in their recesses with the so-called anal glands, which are branching, racemose structures leading deep into the surrounding muscles. It frequently happens that a particularly hard stool, or a seed or other foreign body, will tear or scratch one of the crypts during defecation. This leaves a portal of entry for the numerous bacteria which are always present and a cryptitis ensues. The anal glands at the fundus of the crypt help carry the infection deeper into the perianal tissues. If the invasion of these tissues progresses beyond this point, a variety of paths may be taken. The most common is a direct extension through the substance of the external sphincter into the ischio-

rectal space, where the loose fat breaks down and an ischiorectal abscess develops.

If, on the other hand, the infection from

with the rectum, and it is possible for the abscess to be drained without leaving a fistula-in-ano. Intracutaneous and sub-
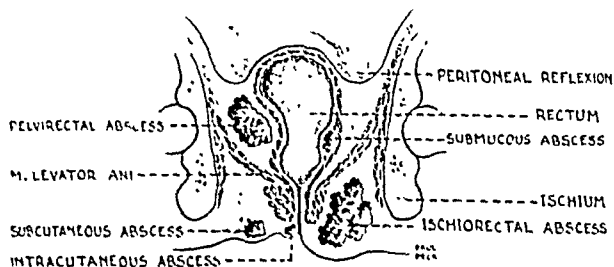
Fig. 1. Section through the pelvis showing location of abscesses mentioned in the text.
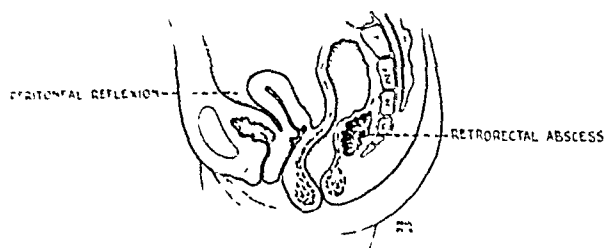
Fig. 2. Sagittal section through the pelvis, showing location of a retrorectal abscess.

the crypt burrows through into the pelvirectal space, a pelvirectal abscess is found.

This is the pathogenesis of all but the most unusual ischiorectal abscesses and explains why a fistula develops following the incision and drainage of the abscess. The fistula is, in fact, merely the contracted wall of the old abscess cavity. Submucous abscesses are caused by a scratch on the surface of the mucous membrane, admitting bacteria which multiply and produce an infection of the submucosa between the rectal mucous membrane and the circular muscle layer.

Inflammatory processes in the various pelvic structures (seminal vesicles, prostate, etc.) may produce pelvirectal abscesses.

A retrorectal abscess may either proceed from a crypt in the usual manner, or may result from infection of the glands of Gerota, a small group of lymph-nodes located in the postrectal space and draining the rectum. When the infection proceeds through the lymph channels to these glands without making a direct fistulous tract, there may be no macroscopic connection

cutaneous abscesses of the perianal region develop from infections through the skin as in other parts of the body and do not produce fistulas. (Fig. 2.)

SYMPTOMS AND DIAGNOSIS

Superficial abscesses are noted by the patient as a swelling beside the anus, growing progressively larger and more painful. The tenderness, especially in the latter stages, is exquisite, and pain precludes the possibility of sleep. Constitutional reactions are present in the form of fever and malaise. Deep abscesses are more gradual in their onset and do not produce such prompt and obvious signs of inflammation. The patient is prone to complain of a heavy feeling and a dull ache deep in the pelvis, lower part of the back, or even down the thighs. Constitutionally, however, the disturbance may be severe.

Diagnosis is usually simple. Inspection and palpation will reveal the classical signs of inflammation in the ischiorectal cases. When seen early, a finger inserted into the anal canal will produce sharp

pain which will be localized by the patient on one side or the other. With an anoscope in position, the surgeon can, by exploring the crypts, arrive at a diagnosis. Often pus will be seen exuding from the offending crypt. At this stage, the differential diagnosis will be between early abscess and fissure-in-ano; the latter, however, is easily revealed by inspection. If symptoms are present suggesting an abscess in the anorectal region, but no external signs are noted, a finger inserted into the rectum will usually demonstrate a tender, boggy swelling behind or to one side of the rectum, causing the rectal wall to bulge into the lumen. If this bulge is lateral, a pelvirectal abscess is diagnosed, and if posterior, a retrorectal. The point of greatest tenderness in a retrorectal abscess is usually in the midline between coccyx and anus. It is obvious, considering the anatomy (Fig. 1), that a swelling felt by the finger in the rectum must be above the levator.

It frequently happens that an abscess may perforate the levator from the ischiorectal space to the pelvirectal or vice versa.

PROGNOSIS

Fistula-in-ano is to be expected following an abscess about the anorectum. Although cases undoubtedly exist of ischiorectal abscesses which, being drained, heal and do not result in fistulas, such instances are much rarer than was formerly believed. Since nearly all of these abscesses start as an infection in a crypt, they become fistulas as soon as they are drained externally either spontaneously or by surgical means. Although complete healing may temporarily occur, sooner or later there will be found a draining sinus which connects with a diseased crypt at the anorectal line. Most fistulas are obvious at once, but occasionally as long as two to five years may elapse between the original incision of the abscess and the development of a fistula. It is in this type of case that an inadequate follow-up gives basis for the illusion that an abscess has been present and has not been followed by a fistula.

Except in known tuberculous patients, the incidence of tuberculosis in these cases will not be over 2 to 5 per cent. There is no sound basis for the ordinary belief that fistula-in-ano (and by implication the abscess preceding the fistula) is of tuberculous origin in most instances.

TREATMENT

The treatment of any anorectal abscess is wide incision and drainage. No temporizing measure has enough value to be recommended. In cases of ischiorectal abscess, it is wise to open and drain well while the infection is small, and even before the tissues have broken down to form pus and produce fluctuation. Nothing is gained by waiting, except to prolong the discomfort and enlarge the scope of the inevitable operation and the size of its subsequent fistula.

INCISION AND DRAINAGE OF SUBMUCOUS ABSCESS

Through a suitable speculum, the patient being in the prone position with hips elevated, an incision is made over the point of fluctuation and in the longitudinal axis of the bowel. No anesthesia is necessary unless the abscess is quite near the anorectal line, when 5 cc. of 1 per cent novocaine injected into the external sphincter will be found sufficient for analgesia. Less bleeding will be encountered if the incision is performed with the actual cautery or the endotherm. The incision is made for the entire length of the abscess, leaving no pockets at either end. Under no circumstance is a transverse incision to be made. A ½ inch wide gauze strip soaked in dichloramine-T, or a small Penrose drain, may be inserted into the cavity and removed in twenty-four hours. Mineral oil ½ to 1 ounce may be given nightly by mouth. The daily application of a 1 per cent aqueous solution of gentian violet to the wound under direct vision through a proctoscope, will aid in healing.

INCISION AND DRAINAGE OF PELVIRECTAL ABSCESS

The patient is placed in an exaggerated lithotomy position, under general, low spinal, or sacrocaudal anesthesia. An incision is then made with the scalpel, anteroposteriorly, and at least 1 inch from the anal verge, so as to avoid severing any fibers of the external sphincter. If pus is encountered in the ischiorectal space the incision is enlarged as described for ischiorectal abscess. An index finger is then inserted into the rectum, and with the other hand a closed Kelly hemostat is pushed against the inferior surface of the levator ani. Although this muscle, with its covering fascia, is ordinarily rather tough, nevertheless when an abscess is present above the levator, it will be comparatively easy to thrust the closed hemostat through it. While this is being done, the finger in the rectum is used as a guide to prevent the clamp from perforating the bowel. When the abscess is entered, the closed blades of the clamp are opened and the instrument withdrawn. The opening in the levator is then enlarged in the direction of its fibers, and a piece of vaseline gauze introduced into the depth of the wound. An area of skin is excised from the lateral edge of the skin incision in order to provide free drainage; dressings are applied.

Immediately following the return of sensation, the wound area is continuously covered with hot, wet saline dressings. The vaseline gauze drain is removed in twenty-four to forty-eight hours, and the wound is opened daily to its furthermost depths with the gloved finger or a glass rod dipped in 1 per cent gentian violet. This is a much less painful maneuver than packing and unpacking with iodoform gauze. The bowels are permitted to move when they will. If no movement has taken place by the third day, a mild laxative or a low saline enema is administered. The patient is allowed out of bed about the third day; after this time the wet dressings are discontinued and hot sitz baths substituted. A sitz bath is ordered following each bowel movement and three times a day. Each bath lasts for ten minutes and is as hot as can comfortably be borne (105 to 110°F.). The patient is usually discharged on the fourth or fifth day and dressed daily in the office thereafter.

INCISION AND DRAINAGE OF RETRORECTAL ABSCESS

The patient is anesthetized and placed in an exaggerated lithotomy position. An incision is made in an anteroposterior direction behind the anus and about one inch to one side of the midline. If desired, the incision may be made directly in the midline, but in this case it is necessary to injure the anococcygeal ligament. The dissection is continued by the method of thrusting ahead a closed hemostat and opening it, or by blunt dissection with the gloved finger, proceeding always posteriorly and toward the midline in the direction of the retrorectal space. When the abscess cavity is reached and the pus is discharged, the opening is enlarged so as to give adequate drainage, the lateral edges of the skin wound are excised to prevent premature healing, and a strip of vaseline gauze or a Penrose drain is inserted. Dressings are then applied.

The after-care is the same as for pelvirectal abscess (vide supra).

INCISION AND DRAINAGE OF INTRACUTANEOUS AND SUBCUTANEOUS ABSCESSES

In the anorectal region, these abscesses, which are not primary at the anorectal line, are rare, and usually small. For drainage, cruciate incision is made and the four quadrants of skin thus formed are trimmed away, thus uncapping the entire abscess cavity. Vaseline gauze is applied. The wound is then dressed daily and will be found to heal readily by granulation from the bottom.

INCISION AND DRAINAGE OF ISCHIORECTAL
ABSCESS

The patient is anesthetized, (using a general, low spinal, or sacrocaudal anesthesia), and is placed in an exaggerated lithotomy position. By palpation, the point of maximum fluctuation is determined. An incision is then made into the fluctuating area as near to the anus as possible, but at least 1½ inches from the anal verge, in order to avoid injury to the external sphincter fibers. The incision is made in a vertical direction, and should be about 2 inches long. After the skin has been cut, a closed hemostat is plunged through the subcutaneous tissue into the abscess cavity, opened and withdrawn. The finger is now inserted and the cavity explored. With the knowledge thus gained, the incision is lengthened anteriorly or posteriorly or both, sufficiently far so that the entire extent of the abscess is opened. All loculi within the cavity are broken down with the finger, taking care not to use so much force that normal tissues are invaded. The *lateral* edge of the skin wound is now excised as far as is necessary to remove the roof of the cavity. If the abscess is large enough, this maneuver will practically uncap the whole ischiorectal fossa. The resulting wound should gape widely at the skin level and grow progressively smaller toward the depths of the wound, thus permitting free drainage. Vaseline gauze is lightly laid into the wound and gauze dressings applied.

The postoperative care is the same as that laid down for pelvirectal abscess.

Comment. The question often arises as to whether an attempt should be made at the time of operation upon the abscess, to locate and remove the internal opening in the crypt, thus curing the fistula before it develops. It is the opinion of the author that in the very occasional instance of a small abscess, superficial to the sphincter, near the anus and with an obvious internal opening, such may be done. In the great majority of cases, however, it is far better to drain the abscess and leave the fistula until later. By so doing, several dangers and difficulties are avoided: The danger implicit in opening clean areas in the presence of virulent pus, the difficulty of working accurately in an anatomy distorted by swelling and edema, the danger of tearing due to friability of the tissues and the danger of severing the sphincter muscle before it has a firm attachment of scar tissue. Generally speaking, a period of six to twelve weeks should elapse between drainage of the abscess and fistulectomy.

SUMMARY

1. Anorectal abscesses are classified according to their anatomical location, and the relevant anatomy is described.

2. The pathogenesis of anorectal abscesses is given.

3. Diagnosis and prognosis are discussed, and the probability of fistula following anorectal abscess explained.

4. Operative treatment for each of the various types is described in detail.

SYMPTOMATIC PILONIDAL CYST

OPERATIVE TREATMENT

Captain Milton N. Camp, m.c. and   Captain Nicholas Polites, m.c.
Assistant Chief of Surgical Service, Station Hospital   Ward Surgeon, Station Hospital

CAMP POLK, LOUISIANA

SYMPTOMATIC pilonidal cysts are cause for frequent admissions on the surgical service in the Station Hospital, Camp Polk, Louisiana. Their appearance is explained by the fact that our soliders for the most part fall within what is known as the "pilonidal age group." They are male adults usually between twenty and thirty years of age and are daily participating in strenuous army activity. During the past twelve months fifty such patients have been operated upon and the pathological condition has varied from a single draining sinus to a previously operated cyst with sinuses. (Figs. 1 and 2.)

TREATMENT

We believe that symptomatic pilonidal cysts should be treated surgically with excision of the pathological condition. The resulting wound heals either by primary intention, granulation followed by epithelization, or a combination of the two, depending upon the manner in which the patient is treated. In our series all types of closure have been used. Excision of the cyst has been followed by (1) no closure, (2) partial closure, or (3) complete closure.

Surgical measures are important in the treatment of the pilonidal cyst but should follow an adequate preoperative preparation. The soldier that presents himself with a pilonidal abscess or a grossly infected draining sinus is not ready for excision of his cyst until all gross evidence of infection has been removed. All cases are put on hot saline sitz baths for fifteen to twenty minutes two to three times daily. The patients are observed daily to make sure that there is always adequate drainage. If an abscess is present, incision and drainage is indicated and then the sitz baths are started. After all purulent drainage and tissue induration has disappeared, surgical excision of the pilonidal cyst is in order. It probably would be advisable to delay the surgery two or three months but this has not proved practical in the army. The patients who have been returned to duty at the time their infection cleared up with instructions to return later for surgery, have usually had another flare-up of their cysts and we have seen them in the same condition as on previous admission, or they are transferred to another camp where the condition becomes symptomatic before they again reach a hospital.

At the beginning of the pilonidal series in this hospital, the general tendency was to leave excised pilonidal wounds open and allow healing to take place by granulation and epithelization. However, a few wounds were partially closed. Neither of these methods gave results that we thought were satisfactory, so the practice of complete closure of the wound was started. With the latter, our results were greatly improved and for the past four months this has been the procedure followed, with one exception. In this one case it was necessary to excise most of the sacrococcygeal ligaments which would have prevented complete closure without leaving undesirable dead space. In this same case there was much wound contamination because the cyst was ruptured during the excision.

Our patient with a pilonidal cyst receives a saline enema the night preceding and the morning of operation. One to two hours before coming to surgery, the patient's sacrococcygeal region is shaved, scrubbed

with green soap and water, painted with tincture of merthiolate and covered with a sterile towel. Preoperative medication con-

FIG. 1. Typical pilonidal sinus (Case No. 41 in series).

FIG. 2. Pilonidal cyst following incision and drainage (Case No. 35 in series).
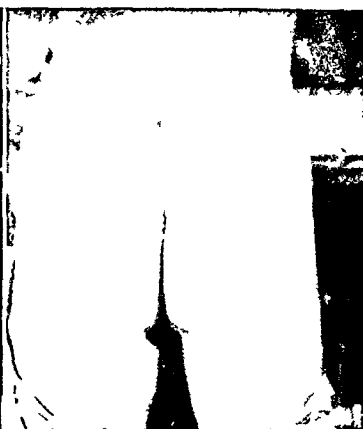
FIG. 3. One hundred per cent healing by primary intention of completely closed pilonidal wound (Case No. 37 in series).

sists of morphine sulfate given hypodermically and barbiturates in some form orally. The anesthetic drug generally used is 120 mg. of spinal procaine. For operation the patient is placed on the table in a prone position and after lowering the lower half of the table about 10 degrees, 3 inch lateral traction adhesive straps are applied to each buttock and fastened beneath the table. This makes the skin tense thereby aiding in minimizing the width of the excision wound and helping to prevent undermining of the skin. The operative site is further prepared with applications of ether and tincture of merthiolate. The pilonidal cyst is distended under moderate pressure with a sterile solution of 1 per cent methylene blue solution with the amount used depending upon the size of the cyst. A 2 cc. syringe is used to which is attached the hub portion of a hypodermic needle. The methylene blue solution furnishes an excellent guide to follow in excising the cyst. The excision of the desired area is performed by making a more or less vertical elliptical excision which is usually extended down to the sacrococcygeal ligaments. Great care is taken not to cut into the cyst and thereby contaminate the field. To remove all

pathological disorders it is not infrequent that portions of the ligaments have to be excised. There have been no removals of the

coccyx. Hemostasis is obtained by using hot saline packs and ties of plain No. 0 catgut or cotton No. 60. At this point the lateral traction adhesive is removed from the buttocks.

With primary wound closure being the desired procedure three to five transverse retention sutures of black silk No. 8 are placed beneath the sacrococcygeal ligaments. Another check is made to see that all hemorrhage is controlled as blood oozing would predispose to a wound breakdown. Sulfanilamide powder is sifted into the wound and into each layer of the closure. A total of 5 to 8 Gm. of the drug is generally used. A row of interrupted chromic No. 1 catgut or cotton No. 20 sutures are placed in the lower portion of the wound again passing beneath the sacrococcygeal ligaments. We wish to emphasize that these sutures are placed below these ligaments to eliminate dead space. The subcutaneous tissues are closed with interrupted plain No. 0 catgut or cotton No. 30 and the skin edges are approximated with end on mattress sutures of black silk No. 1. The retention sutures are tied over a 3 to 5 inch rolled dry sponge which covers the closed wound. Further

CASE HISTORIES

No.	Date Admitted	Age	Duration since Symptoms First Noticed	Admission Pathology	Incision and Drainage	Preoperative Time	Type Wound Closure	Post Operative Time	Total* Hospital Days	Comments
1	6/17/41	26	2 years	Draining sinus.	No	6 days	Partial	37 days	43 days	Complete healing.
2	6/24/41	23	3 years	Draining sinuses.	No	2 days	Open	66 days	68 days	Complete healing.
3	6/27/41	23		Draining sinuses.	No	11 days	Open	75 days	86 days	Complete healing.
4	6/30/41	26	2 mos.	Draining sinuses.	No	8 days	Partial	185 days	193 days	Before complete healing obtained scar tissue excised and wound grafted.
5	9/11/41	19	4 days	Draining sinus.	Yes	18 days	Partial	42 days	60 days	Complete healing.
6	9/30/41	23	2 weeks	Draining sinuses.	No	7 days	Partial	68 days	75 days	Non-healing wound curetted before complete healing obtained.
7	10/3/41	25	2 years	Draining sinuses.	No	6 days	Open	38 days	44 days	Complete healing.
8	10/23/41	21	8 mos.	Draining sinuses.	Yes	4 days	Open	46 days	50 days	Complete healing.
9	10/23/41	26	6 mos.	Abscess.	Yes	7 days	Open	153 days	160 days	Before complete healing obtained scar tissue twice excised.
10	11/19/41	22	2 weeks	Abscess.	Yes	6 days	Open	57 days	63 days	Returned two weeks following discharge with ulcer at site of healed wound. Discharged 11 days later healed.
11	12/19/41	28		Abscess.	Yes	11 days	Open	58 days	69 days	Complete healing.
12	1/5/42	25		Draining sinus.	No	1 day	Complete	20 days	21 days	100% primary intention. Incision and drainage 3 weeks prior to admission. Returned for excision.
13	1/26/42	22	2 years	Draining sinus.	No	1 day	Complete	12 days	13 days	100% primary intention.
14	2/6/42	31	3 years	Draining sinus.	No	10 days	Complete	36 days	46 days	98% primary intention.
15	2/9/42	23	2 mos.	Cellulitis.	No	8 days	Complete	64 days	72 days	50% primary intention.
16	2/15/42	22	3 mos.	Draining sinuses.	Yes	32 days	Complete	64 days	96 days	80% primary intention
17	2/25/42	22		Draining sinus with previously excision scar wound.	No	13 days	Open	43 days	56 days	Complete healing. Previous operation Sept. 1941 at another Army Camp.
18	3/3/42	25	3 days	Draining sinus.	No	14 days	Complete	15 days	29 days	100% primary intention.
19	3/9/42	23	7 days	Draining sinus.	No	7 days	Complete	19 days	26 days	100% primary intention.
20	3/9/42	22	3 mos.	Abscess.	Yes	15 days	Complete	14 days	29 days	100% primary intention.
21	3/11/42	25	4 days	Draining sinus.	No	8 days	Complete	15 days	23 days	100% primary intention.
22	3/13/42	22	1 week	Abscess.	Yes	10 days	Complete	12 days	22 days	100% primary intention.
23	3/21/42	23	2 years	Abscess.	Yes	23 days	Complete	74 days	97 days	50% primary intention.
24	3/23/42	32	5 days	Abscess.	Yes	28 days	Complete	24 days	52 days	100% primary intention.
25	3/24/42	26	3 weeks	Cellutitis.	No	9 days	Complete	64 days	73 days	100% primary intention. Healed completely in 28 days post operatively, but had skin slough at spinal site which necessitated the stay.
26	3/25/42	24	2 weeks	Draining sinus.	No	8 days	Complete	64 days	72 days	60% primary intention.
27	3/27/42	20	2 years	Draining sinus.	No	7 days	Complete	44 days	51 days	98% primary intention.
28	3/31/42	22	8 years	Abscess.	Yes	23 days	Complete	†	†	0% primary intention. ½" superficial granulating wound remains.
29	4/3/42	24	7 days	Draining sinus.	No	20 days	Complete	39 days	59 days	75% primary intention.
30	4/9/42	30	1½ years	Draining sinus.	No	1 day	Complete	16 days	17 days	100% primary intention. Sitz baths advised 1 week prior to hospitalization.
31	4/10/42	28		Draining sinus.	Yes	3 days	Complete	30 days	33 days	75% primary intention.
32	4/27/42	23	3 weeks	Abscess.	Yes	17 days	Complete	46 days	63 days	75% primary intention.
33	4/27/42	23	3 days	Abscess.	Yes	14 days	Complete	24 days	38 days	100% primary intention.

CASE HISTORIES (*Continued*)

No	Date Ad- mitted	Age	Duration since Symptoms First Noticed	Admission Pathology	Incision and Drain- age	Preop- erative Time	Type Wound Closure	Post Operative Time	Total* Hospital Days	Comments
34	4/27/42	25	1 year	Draining sinus	Yes	14 days	Complete	†	†	80% primary intention. ⅜" superficial granulating wound remains
35	5/1/42	35	2 years	Draining sinus	Yes	31 days	Complete	†	†	100% primary intention 20 days postoperative
36	5/2/42	26	5 years	Draining sinuses	No	4 days	Complete	19 days	23 days	100% primary intention History of at least 15 inci- sion and drainages in the past
37	5/7/42	24	1 year	Draining sinus	No	11 days	Complete	†	†	100% primary intention. Very large closure wound See figure
38	5/13/42	33	2 years	Draining sinus	No	7 days	Complete	18 days	25 days	100% primary intention.
39	5/20/42	22	2 years	Draining sinus	No	21 days	Complete	†	†	100% primary intention. 20 days postoperative
40	5/27/42	26	4 mos	Abscess	Yes	22 days	Complete	†	†	100% primary intention 12 days postoperative
41	5/29/42	22	3 years	Abscess	Yes	15 days	Complete	†	†	75% primary intention
42	6/2/42	27	3 weeks	Abscess	Yes	8 days	Complete	†	†	100% primary intention 20 days postoperative
43	6/2/42	25	3 days	Draining sinus	No	9 days	Complete	†	†	100% primary intention 19 days postoperative
44	6/4/42	24	2 years	Draining sinus	No	7 days	Complete	†	†	75% primary intention
45	6/8/42	25	2 days	Abscess	Yes	10 days	Complete	†	†	100% primary intention 12 days postoperative
46	6/9/42	34	3 days	Draining sinus	No	6 days	Complete	†	†	100% primary intention 15 days postoperative
47	6/10/42	33	2 days	Draining sinus	No	5 days	Complete	†	†	100% primary intention 15 days post-operative.
48	6/13/42	31	1 mo	Abscess	Yes	9 days	Complete	†	†	100% primary intention. 8 days post-operative
49	6/18/42	20	1 year	Draining sinus	No	4 days	Open	†	†	Cyst ruptured into during operation so closure not thought advisable
50	6/18/42	25	5 mos	Draining sinus	No	4 days	Complete	†	†	100% primary intention 8 days post-operative

1 Average pilonidal cyst age—25 years
2 Average time symptomatic pathology present—1 year
3. 50% of the cases had had disturbances prior to army service
4 Average pre-operative treatment time—10 days
5. Average total hospitalization with no closure of wound—74 days (8 completed cases).
6 Average total hospitalization with partial closure of wound—93 days (4 completed cases).
7 Average total hospitalization with complete closure of wound—45 days (22 completed cases).
8. 64.8% of the cases with complete closure obtained 100% primary intention.
* Patient hospitalized until condition permits return to army duty
† Denotes patient still in hospital

dry dressings are applied and the buttocks are firmly strapped together.

Postoperative treatment consists of the foot of the bed elevated for six hours to prevent postspinal headache, liquid diet for five days, and absolute bed rest with frequent change of position for ten to twelve days plus usual symptomatic meas- ures. Bowel movements are discouraged and generally are not a disturbing factor. A saline enema is given on the sixth post- operative day. This is the first day the dressing is changed following operation and the time when the skin sutures are re-

moved. The retention sutures are left in place eight to ten days if possible. The patient usually walks freely about the ward on the twelfth to fourteenth day. One case (a nurse) has, however, been discharged to duty as early as the twelfth day. (Fig. 3.)

A precautionary measure taken at the time of discharge from the hospital is to request the soldier's Commanding Officer to give him duty for two weeks where motorcycle driving, truck driving and excessive walking can be avoided. This is not an unusual request, for following any

type of surgery a soldier should be allowed time to again adjust himself to life in the field. There have been no cases to return with recurrent cysts in this series. One patient was placed on guard duty immediately following hospitalization and there was a slight breakdown of his previously healed wound. We believe that excessive walking was the causative factor. After eleven days of additional hospitalization, the soldier was discharged to duty as cured. (Table 1.)

SUMMARY

1. A series of fifty operations on symptomatic pilonidal cysts is reported.

2. Preoperative care of the pilonidal cysts to minimize infection is accomplished by repeated hot sitz baths.

3. Our operative method of choice is excision followed by complete wound closure.

4. To minimize the width of excision wounds and aid in preventing undermining of the skin, lateral traction of the buttocks is maintained during operation.

5. Complete hemostasis of excised pilonidal wounds is obtained before a closure is made.

6. Dead space is eliminated in the closure by placing deep sutures and retention sutures beneath the sacrococcygeal ligaments.

7. Infection of the closed pilonidal wound is usually prevented by sifting powdered sulfanilamide throughout the wound closure.

THE SUSPENSORY MUSCLE OF THE DUODENUM

J. C. HALEY, M.D. AND J. K. PEDEN, M.D.

Associate Professor of Anatomy, Baylor University
College of Medicine

Assistant Professor of Anatomy, Baylor University
College of Medicine

DALLAS, TEXAS

THE primary purpose of this study has been to determine the manner of attachment of the suspensory muscle,

ANATOMY OF STRUCTURES INVOLVED

The duodenum consists of the first ten inches of the small intestine and is held
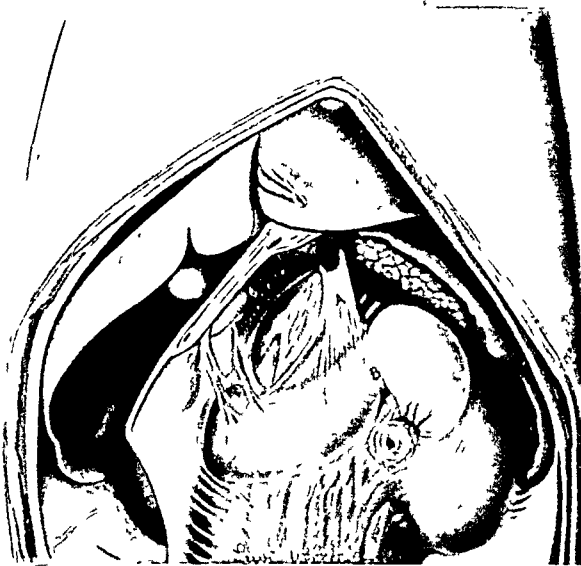
FIG. 1. Suspensory muscle of duodenum in position in an adult. A, suspensory muscle of duodenum; B, duodenojejunal flexure.

FIG. 2. Suspensory muscle of duodenum in position in a newborn infant. A, suspensory muscle of duodenum; B, duodenojejunal flexure.

or ligament, of the duodenum and to attempt to deduce the probable effect of this attachment and the contraction of the muscle upon the size of the angle at the duodenojejunal flexure. Incidentally, we have attempted to determine if there is a considerable percentage of cases in which muscle fibers can be demonstrated in this structure.

Some clinicians have contended that this structure is attached directly to the flexure and as a result of this attachment, the size of the angle at the flexure is decreased during contraction of the muscle and movement of the gut, with consequent tendency toward causing partial obstruction at this point.

against the posterior abdominal wall, largely behind the peritoneum, in such a manner as to describe a C-shaped curve with the distal end of the "C" turning sharply forward and downward to form the duodenojejunal flexure. For descriptive purposes it is usually divided into four parts. The first part about two inches in length, begins at the pyloric end of the stomach at the level of the first lumbar vertebra and extends to the right and slightly backward to the neck of the gallbladder where it turns rather sharply downward to become the second part. The second part, about three inches in length, runs downward to the level of the third lumbar vertebra, where it bends abruptly
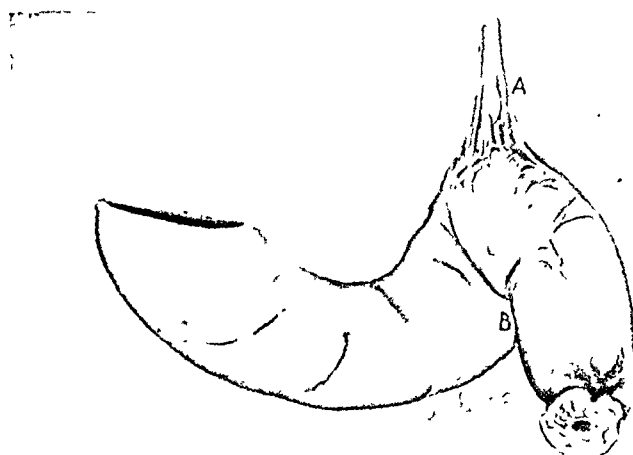
FIG. 3.
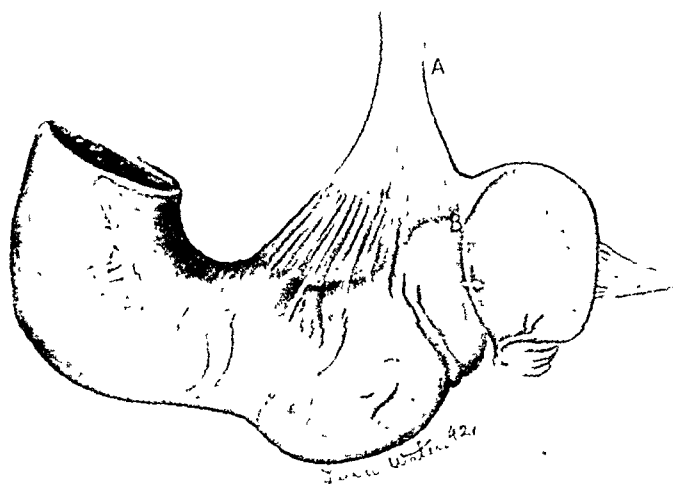
FIG. 4.
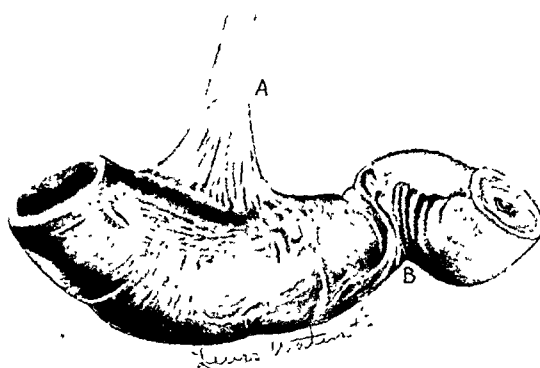
FIG. 5.

FIG. 3. Suspensory muscle of duodenum after removal from the body. A, suspensory muscle inserted solely into the duodenojejunal flexure, B.

FIG. 4. Suspensory muscle of duodenum after removal from the body. A, suspensory muscle inserted into the duodenojejunal flexure, B, and into the fourth and third parts of the duodenum.

FIG. 5. Suspensory muscle of duodenum after removal from the body. A, suspensory muscle inserted into fourth and third parts of the duodenum with no attachment to the duodenojejunal flexure, B.

to the left to become the third part. The third part, nearly four inches in length, runs almost horizontally to the left, with a

ance of this structure in an adult left in position after dissection. Figure 2 shows it similarly in a newborn child. Figures 3, 4
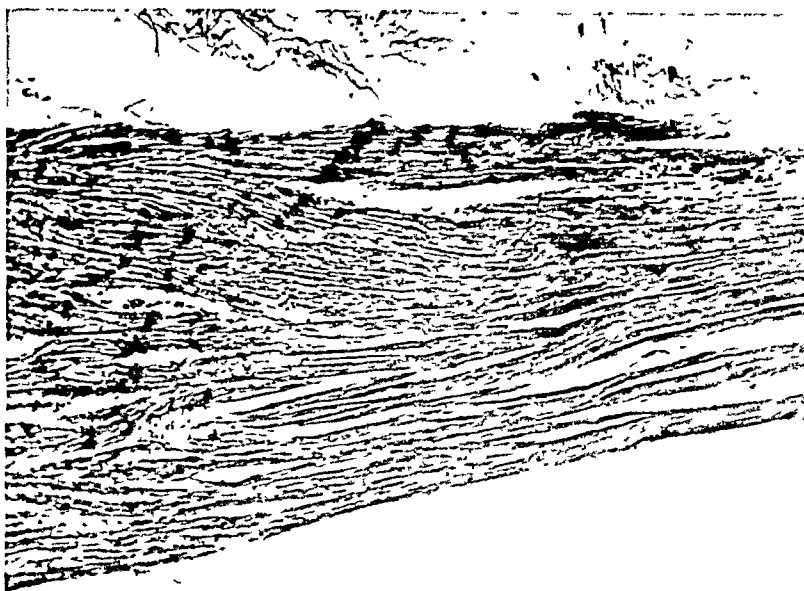
FIG. 6. Microphotograph showing section of suspensory muscle of duodenum as seen under low magnification.

slight inclination upward, to a point just to the left of the median plane, where it turns upward to become the fourth part. The fourth part, a little more than an inch in length, runs upward to the level of the second lumbar vertebra, where it turns sharply forward and downward as the duodenojejunal flexure.

The suspensory muscle of the duodenum takes origin from the right crus of the diaphragm and from the dense fibrous tissue around the celiac artery. From this origin it extends downward behind the pancreas, the splenic vein, and in front of the left renal vein to be inserted into the duodenum. The location of this insertion is the chief object of this study.

PROCEDURE

Gross Study. We dissected this structure in seventy-seven cadavers, thoroughly cleaning it from its origin to its insertion, noting carefully its precise attachment to the gut, and also noting presence or absence of muscle fibers as could be detected grossly. Figure 1 shows the gross appear-

and 5 show it after its removal from the body.

Microscopic Study. We took sections from forty specimens, without any attempt at selection, to be examined microscopically for presence or absence of muscle fibers. Twenty of these were taken from the group in which muscle fibers could be demonstrated grossly, and twenty from the group in which muscle fibers could not be definitely identified grossly. Figure 6 shows a photomicrograph of one of these sections under low magnification and Figure 7 shows a photomicrograph of one under high magnification.

RESULTS

Gross Findings. In four (5.2 per cent) of our specimens the fibers of the suspensory muscle were attached solely to the flexure, no fibers being attached to the fourth and third parts of the duodenum; in forty-seven (61 per cent), the fibers were attached to the flexure and to the fourth and third parts of the duodenum; and in twelve (15.6 per cent) the fibers

were attached solely to the fourth and third parts of the duodenum, no fibers being attached to the flexure.

at the flexure upon movement of the gut or contraction of the suspensory muscle, thus causing a partial obstruction at this
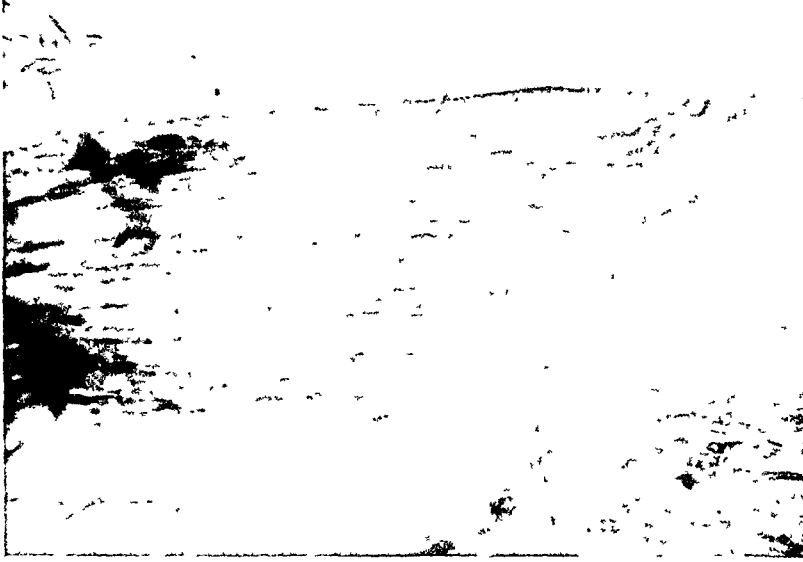
FIG. 7. Microphotograph showing section of suspensory muscle of duodenum as seen under high magnification.

In fourteen (18.2 per cent) of our specimens neither muscle nor ligament could be demonstrated, the fibers apparently having blended with the surrounding fibrous tissue.

In twenty-four (31.2 per cent) of our specimens there appeared, from gross examination, to be muscle fibers present; and in thirty-nine (50.6 per cent) one could not determine, from gross examination, whether or not muscle fibers were present.

Microscopic Findings. In eighteen of the sections taken from the twenty cases in which muscle fibers could be demonstrated grossly, muscle fibers could be demonstrated microscopically; and in sixteen of the sections taken from the twenty cases in which muscle fibers could not be definitely identified grossly, muscle fibers could be demonstrated microscopically.

DISCUSSION

By referring to Figure 3, one can see that in the four cases (5.2 per cent) in which the muscle or ligament was attached solely to the flexure there might be a tendency to reduce the size of the angle

point. But by referring to Figures 4 and 5, it will be seen that in the fifty-nine cases (76.6 per cent) in which this structure is attached to the fourth and third parts of the duodenum, there would seem to be a tendency to prevent any change in the size of the angle at the flexure and thus to aid in prevention of constriction of the gut at this point.

Since there is such a scarcity of reports in the literature on this structure, there seems to have been very little investigation of it recently. The textbooks in gross anatomy apparently convey the idea that in adults there are rarely to be found any muscle fibers in it. But it will be noted that in our series there were muscle fibers present in thirty-four of the forty specimens examined microscopically. All of these cases were adults and probably all of them were over thirty years of age.

In the description of this muscle in Morris' Human Anatomy[4] it is hinted that there might be a few skeletal muscle fibers present, these being derived from the crus of the diaphragm. All other authors are noncommittal on this point. We made a

particular study of this and found only nonstriated muscle fibers present.

Although we have been unable to find any reports of a study of the nerve supply of this muscle, it might be assumed that since its muscle fibers are similar to that of the gut, its nerve supply would be the same as that of the gut. If this be true, its contraction might be synchronous with the peristaltic movements of the duodenum. In view of the manner of insertion of the muscle into the duodenum, as found in most of our cases (76.6 per cent into the fourth and third parts of the duodenum), it would seem that its contraction has a *tendency to increase the size of the angle* at the flexure during movement of the duodenum, thus preventing obstruction at the flexure.

The authors wish to thank Mr. Lewis Waters, Instructor in Medical Art, Baylor University,

REFERENCES

1. CROSTHWAIT, W. L. Duodenal obstruction. *Texas Surgical Society*, pp. 93–97, 1935.
2. CUNNINGHAM, D. J. Text Book of Anatomy. 7th ed., pp. 424 and 603. New York, 1937. Oxford University Press.
3. GRAY, H. The Anatomy of the Human Body. 24th ed., pp. 1190–1191. *Philadelphia*, 1942. Lea and Febiger.
4. MORRIS, Human Anatomy. 10th ed., p. 1311. Philadelphia, 1942. The Blakiston Company.
5. PIERSOL, GEORGE A. Human Anatomy, 9th ed., p. 1645. Philadelphia, 1930. J. B. Lippincott Company.
6. TREVES, F. Applied Anatomy. 9th ed., p. 440. Philadelphia, 1934. Lea and Febiger.

VENOGRAPHY OF LOWER EXTREMITY*

A NEW TECHNIC—PRELIMINARY REPORT

EMILE ZAX, M.D.

Assistant Attending Surgeon, Jefferson Davis Hospital.

HOUSTON, TEXAS

THE film labelled "James Matthews" demonstrates a venogram of the deep vessels of a normal lower extremity. saphenous vein. This was obtained using exactly the same procedure as in the "James Matthews" case and would usu-
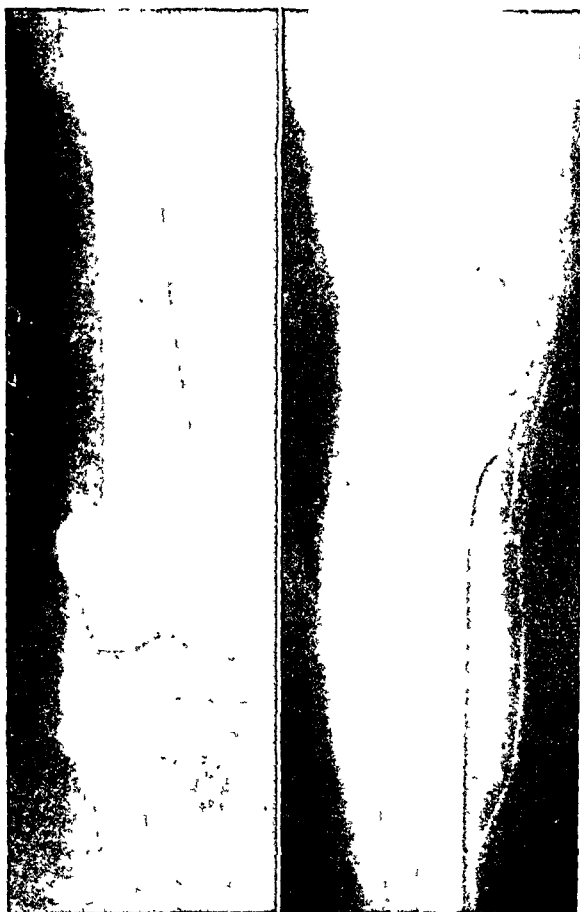
FIG. 1. Venogram showing popliteal and femoral vein.

FIG. 2. Venogram showing great saphenous vein.

This was obtained by the method commonly in use—injection of diodrast into the small saphenous vein exposed through an incision posterior to the lateral malleolus. The film labelled "Rosie Denison" demonstrates a venogram of the great ally be interpreted as a blockage of the popliteo-femoral vein. The finding was very unexpected because there was no history nor signs of any vascular disturbance in the limb, and the venogram had been done to demonstrate the deep

FIG. 3. Apparatus to compress great saphenous vein at knee

FIG. 4. Apparatus in place, patella is outlined.
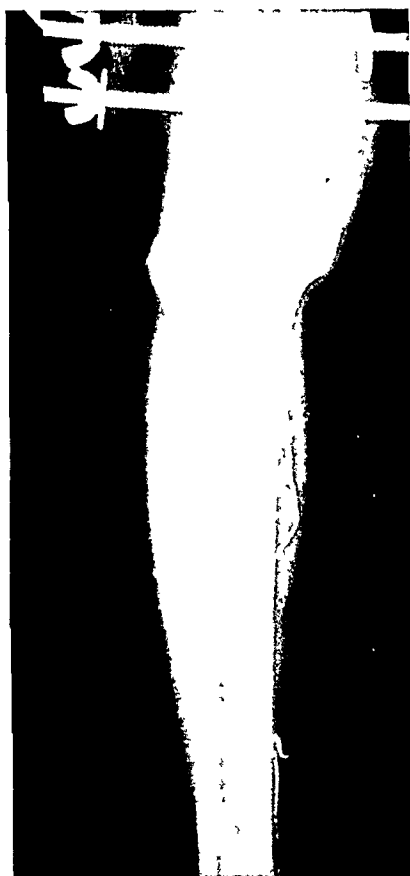
veins of a normal lower extremity. The explanation regarded as the most likely was that in this case there was less resistance to the flow of the injected solution in the great saphenous than in the small saphenous vein causing the solution to go into the saphena magna through the communicating veins of the distal leg.

To obviate such happening when deep vein blockage is suspected and also to do away with the necessity of cutting down on the small saphenous vein, I devised the crude apparatus illustrated separately and in position at the knee. It consists of two blocks of wood, each six inches by six inches by one-half inch, well padded with felt as illustrated and connected by two iron bars eight inches in length and threaded for three inches at each end. It is slipped over the leg until the anterior bar is at the midpatella region and then tightened until the patient states it is very tight. The purpose of this is to compress the great saphenous vein at the medial side of the knee but not the small saphenous vein in the popliteal area. Such being the case, diodrast injected in any vein of the

foot or ankle region should have to pass this area by going through the deep veins or the small saphenous vein. If the deep vein should be blocked due to a pathological process, a detour around the compressed area of saphena magna or a failure of the dye to go farther should be expected.

In the one case illustrated using this apparatus, the diodrast was injected into a vein on the dorsum of the first metatarsal bone. It can be seen that in the area of the knee the diodrast is in the popliteal vein, and there is none in the saphena magna. (This patient had no vascular disturbance, the diodrast being given for an intravenous urogram.) The idea of blocking the great saphenous vein in order to demonstrate the deep veins seems justified in this one case. The ease of venipuncture rather than the operative procedure and sterile set-up necessary for cutting down on the small saphenous vein also recommends a trial of the procedure to both physician and the patient. The apparatus described can probably be greatly improved as the method is put to further use.

THE USE OF SULFATHIAZOLE VASELINE OINTMENT*

VOIGT MOONEY, M.D. AND F. A. TAYLOR, PH.D.

PITTSBURGH, PENNSYLVANIA

THE use of vaseline gauze strips on operative incisions, before dressings are applied, has been a tried surgical
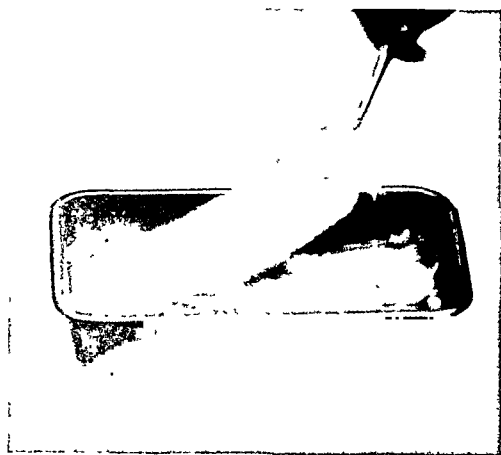
FIG. 1. Pan and 5 per cent sulfathiazole vaseline gauze.

procedure. Wishing to contribute more security to the postoperative area, I have used, with success, the following plan. Two-inch gauze strips are planted in a pan of 5 per cent sulfathiazole vaseline ointment. The pan is sterilized in the autoclave for forty-five minutes at twenty pounds pressure.†

The effect of steam sterilization on the sulfathiazole suspension was examined by placing a sample of the ointment in the sterilizer along with the impregnated gauze so that both were similarly heated. That the control sample was not in contact with the dry gauze was considered unimportant. After heating, the petrolatum of the control was dissolved in petroleum ether. The sulfathiazole was filtered on a fritted glass crucible and washed thoroughly with the same solvent.

Without further purification, the recovered sulfathiazole melted at 197–199°C. while the original material melted at 198–200°C. in comparison (both uncorrected).

Only traces of substances active in the Bratton and Marshall method of determining the sulfanilamides were extracted from the petroleum ether by alkali.

There is thus no evidence of decomposition of the sulfathiazole during sterilization.

CONCLUSIONS

1. The incision heals faster with 5 per cent sulfathiazole ointment gauze.

2. Stitch abscesses are virtually not seen after the use of this vaseline gauze.

3. Sulfathiazole ointment gauze is used successfully as packing in osteomyelitis.

4. There is no evidence of decomposition of sulfathiazole during sterilization.

PRIMARY CARCINOMA OF THE FALLOPIAN TUBE*

Joseph L. McGoldrick, m.d.,
Attending Gynecologist, Brooklyn Cancer Institute

Hyman Strauss, m.d.
Associate Gynecologist, Brooklyn Cancer Institute

AND

John Rao, m.d.
Resident Physician, Brooklyn Cancer Institute
BROOKLYN, NEW YORK

OF 376[1] cases of primary carcinoma of the Fallopian tube reported in the world's literature[1-16] to date, we were able to find only one case which was correctly diagnosed preoperatively.[17] This striking fact emphasizes the need of reviewing the clinical features of the disease so that tubal carcinoma may be considered a diagnostic possibility. Unless we include this in our differential diagnosis, the likelihood of a correct clinical diagnosis is thereby reduced. The purpose of this paper is to emphasize the significance of the symptoms and signs of this clinical entity.

Etiological Considerations. The disease is most frequent in the climacteric years. Cases have been reported, however, as early as eighteen and as late as seventy-three years of age. Its incidence in gynecological malignancies, as reported by various observers, varies from 0.2 to 0.5 per cent. The lesion is bilateral in about one-third of the cases.[18]

Many observers believe that inflammation is a contributory factor in the etiology of tubal malignancy. We incline to the opinion that it bears no causal relation to the disease for the following reasons: (1) If inflammation were a factor, tubal cancer would be much more common. (2) Tubal cancer has been demonstrated to have been present in a large number of cases unasso-ciated with inflammatory lesions. (Vest[19,20] in 132 cases found fifty with no accompanying inflammation.)(3) It is highly probable that in a large percentage of the cases in which inflammation was present it was the result rather than the cause.

PATHOLOGY

A review of morbid anatomical observations indicates the following to be the most probable sequence of events in the pathogenesis of the lesion. The symptoms and course of the disease depend upon which segment of the tube is initially involved. In 70 per cent of the cases the lesion appears to start at the fimbriated end. The fimbriae adhere early and a hydrosalpinx results. With increasing distention of the tube, pain ensues and peristaltic contractions result in extrusion of the tubal contents into and through the uterine cavity, producing leucorrhea. The discharge may be serosanguineous or bloody depending upon the extent of hemorrhage within. The relief of tubal distention by periodic discharge causes remission of pain, a noteworthy characteristic of this symptom. In the remaining 30 per cent, the lesion starts in the isthmus. This type of lesion carries the worst prognosis because here, too, tubal occlusion occurs early and with it there is intraperitoneal dissemination of the carcinoma. Rarely, are there tubal

* From the Brooklyn Cancer Institute.

occlusions at both ends, which would confine the lesion to the interior of the tube.

From the standpoint of morphology there are two types, papillary and infiltrating. The former grows into the lumen of the tube and metastasizes comparatively late, the latter grows into the wall of the tube, penetrates the serosa and spreads early intraperitoneally. Like other types of cancer, tubal carcinoma metastasizes by direct extension and through the lymphatics. The lymphatic pathway is usually along the upper margin of the broad and infundibulopelvic ligaments and the ovarian vessels to the iliac, aortic and lumbar nodes. It also may spread via the round ligament and produce inguinal node enlargement. The close interweaving of the lymphatics of the tube with those of the ovary and uterus may cause early extension to these structures.

Metastases in tubal cancer have been reported[21] in the cervix and fundus uteri, ovary, vagina, bladder, kidney, liver, lungs, stomach, large intestine, diaphragm, spleen, skin and supraclavicular nodes.

SYMPTOMATOLOGY

Intermittent lower abdominal pain, relieved by leucorrhea, and a receding palpable adnexal mass are the triad of symptoms that should suggest tubal cancer when making a differential diagnosis. According to Doran,[22] pain was present in 53 per cent of his cases. Since about 30 per cent of tubal carcinoma is bilateral, the pain may also be bilateral. It is cramp-like and lower abdominal in type and may be relieved by vaginal discharge which reduces tubal distention by extrusion of its contents into the uterine cavity. This type of pain with the associated periodic remission is present in lesions in which the fimbria is occluded. Lesions not situated in this area usually produce a constant dull, aching pain.

DIAGNOSIS

A review of the physical findings before operation reveals that from 70 to 85 per cent of the patients were operated upon because of the presence of a palpable mass on pelvic examination. The association of masses with inflammatory disease of the adnexae and their great preponderance as compared with neoplastic masses may exclude this disease entirely as a diagnostic possibility in the mind of the casual examiner. Many cases are overlooked even at operation and diagnosed as hydrosalpinx and simple procedures are performed. At laparotomy, every adnexal mass should be incised and examined, as an apparent hydrosalpinx may conceal within itself a tubal carcinoma and an ovarian cyst may contain an ovarian neoplasm. Frozen section may save the surgeon the embarrassment of a second laparotomy. The presence and type of symptoms depend upon the size of the lesion, pressure on adjacent structures, and the presence or absence of metastases.

At this point, it is important to include a discussion of the use of uterine curettage in the diagnosis of tubal cancer. If a patient has a sanguineous discharge with no demonstrable lesion and a careful uterine curettage reveals a normal or atrophic endometrium, this finding suggests a laparotomy to rule out a tubal cancer. If the endometrium is hyperplastic, a granulosa cell tumor of the ovary is suggested. In either case, a laparotomy is indicated if the patient is operable. The possibility of tubal or ovarian cancer should condemn the practice of radium or x-ray therapy for postmenopausal uterine bleeding without a preliminary curettage and other indicated diagnostic procedures.

We can summarize the avoidable errors in diagnosis as follows: (1) Do not overlook the possibility of tubal cancer in a patient with bilateral adnexal masses. Thirty per cent of all tubal cancer is bilateral. (2) Do not neglect to open a hydrosalpinx immediately after removal. The findings may demand a more radical procedure. (3) Do not be satisfied with a negative curettage in a case of unexplained postmenopausal uterine bleeding. Look further and if

necessary do a laparotomy, as you may find a tubal cancer. (4) Do not overlook constitutional factors. The patient may have a systemic cause for her bleeding.

PROGNOSIS

There is one insurmountable barrier to the early diagnosis of tubal cancer and that is the absence of symptoms in early malignancy of this organ. Of the cases reported, 99 per cent were discovered at laparotomy or at autopsy and the diagnosis was almost invariably made by the pathologist.

Reliable figures for the survival rate in this disease are lacking because of the following reasons: (1) Very few of the cases reported are adequately followed. (2) Autopsy findings are mostly of those patients that come to the clinician in the late stages of the disease so that the antecedent course cannot be accurately determined. (3) The large majority of these cases had exploratory, surgical procedures or palliative radiation therapy which makes the efficacy of treatment doubly difficult to determine.

The following figures on survival periods are given by various authors:

Wharton[23]—3 out of 14 patients were alive 3 years or more, about 21 per cent.

Wechsler[24]—6 out of 200 cases of tubal cancer survived 3 years, about 3 per cent.

Doran—10 out of 40 postoperative cases survived only 6 months

Watkins[25,26]—followed 3 cases. All died within 3 years; several others report 9 cases with survival periods of 3, 5, 8, 19 and 20 years

Graves[27]—quotes Shack and gives 2 per cent as the rate of cure

Of the 8 cases reported by Parsons[28] and well followed, 2 are alive, but treated too recently for satisfactory evaluation; one is lost and 5 are dead; three of the patients who died received x-ray therapy.

Kehrer[29]—in 80 cases found 5 cures, or 6.2 per cent

These figures plainly indicate a very poor prognosis. The general impression, as in most types of cancer, is that the localized and completely resectable lesion holds out good hopes of cure and that radiation therapy,[30] although not curative, may prolong life and contribute materially to the comfort of the patient. We have no grounds for drawing further conclusions.

The accepted method of treatment, therefore, is complete hysterectomy with wide excision of the adnexae and broad ligaments followed by x-ray therapy. Although the efficacy of x-ray therapy has not been proved, we believe the patient should have the benefit of the doubt.

CASE REPORTS

CASE I. L. R., a fifty-four-year old, married, white female was admitted to the Brooklyn Cancer Institute April 22, 1940, complaining of profuse leukorrhea and irregular vaginal bleeding of three months' duration. Previous menstrual history was normal. She was a gravida VII and para V. All labors were difficult; two deliveries were by means of forceps. Her general condition was good with no weight loss or other symptoms.

Vaginal examination revealed a parous introitus, scarred pelvic floor, transverse laceration of the cervix, uterus small and anterior, parametria free, and adnexae slightly thickened. There was a sanguineous leukorrheal discharge. The impression was carcinoma of the corpus uteri. Four days later, a diagnostic curettage revealed small, nontortuous uterine glands lined by low columnar nonsecretory epithelium. No evidence of malignancy was found. One week later, laparotomy showed a pseudohydrosalpinx on the left. A supracervical hysterectomy and a bilateral salpingo-oophorectomy were performed. On section, the left tube contained a large soft mass within its lumen and was closed at the isthmic and fimbriated ends.

Macroscopic examination revealed the left tube to be 8 cm. in length. The isthmic end was narrow, the fimbriated end 1 cm. in diameter and completely closed. On cut section, the wall of the distal 2 cm. was thickened and consisted of a firm, yellowish white tissue which was thrown into what appeared to be numerous large plicae. The lumen contained no fluid. The right tubal wall was thin, the fimbria patent and the lumen empty. Both ovaries

were atrophic. The uterus was small and showed no gross evidence of neoplasm.

Microscopic examination showed the section

FIG. 1. Case 1. Left Fallopian tube primary anaplastic carcinoma. 50 X.

through the mass at the fimbriated end of the left tube to consist of broad sheets of atypical cells, groups of which widely invaded all the tissues of the wall. No muscularis was recognizable, this having been replaced by dense, hyaline, poorly cellular fibrous tissue, heavily infiltrated with lymphocytes. The individual tumor cells were large, having poorly defined cell boundaries, pale pink cytoplasm, and round or ovoid vesicular nuclei with prominent nucleoli. Regular mitotic figures were common. In the uninvolved portions of the tube the plicae were broadened and interconnected to form pseudoglands. The fibrous tissue was increased and infiltrated with numerous lymphocytes, plasma cells, polymorphonuclear leukocytes and numerous hemosiderinladen phagocytes. The inflammatory infiltrate extended into the muscularis. Sections through the right tube, both ovaries and uterine wall presented no tumor either primary or metastatic. *Diagnosis:* Anaplastic carcinoma of the left Fallopian tube.

Convalescence was uneventful. Four months later, a cycle of high voltage Roentgen therapy was given prophylactically to the pelvis. January 19, 1942, twenty-one months later, the patient gained weight, had no complaints, and pelvic examination was negative for residue or recurrence.

CASE II. B. H., a forty-four-year old, married negress, was admitted to the hospital, on May 20, 1940, because of a serosanguineous vaginal discharge of seven months' duration. She complained of no abdominal pain. Previous menstrual and obstetrical history was non-contributory. Her past history was irrelevant except for acute salpingitis at the age of twenty-two. Vaginal examination revealed an enlarged nodular fibroid uterus. A hysterosalpingo-oophorectomy was performed. The uterus was moderately enlarged by multinodular fibroids, undergoing hyaline degeneration. The endometrium was atrophic. A hydrosalpinx with a papillary adenocarcinoma showing degenerative and inflammatory changes was present in the left tube. Only chronic salpingitis was found in the right. Both ovaries showed vascular sclerosis and corpora albicantia but no malignancy.

Four months later she was referred to the Brooklyn Cancer Institute where a cycle of high voltage x-ray therapy was given. A year later she was operated upon for the removal of a cyst of Gartner's duct. On January 22, 1942, twenty months later, she was free from evidence of recurrence and had gained twenty-five pounds.

CASE III. M. D., a forty-two-year old, married, white female was admitted to the hospital June 3, 1940, with a complaint of "irregular menstrual bleeding of six months' duration," and bilateral lower abdominal pain radiating to the back. Menstrual, obstetrical and past history were nonessential. Vaginal examination showed a large, fixed mass deep in the pelvis filling the hollow of the sacrum and a soft mass in the region of the left adnexa. At laparotomy, the findings were: "Uterus and

adnexa bound down by fine adhesions to pelvic peritoneum and intestine. Right tube and ovary the seat of a large cystic, adherent mass deep

Fig. 2. Case 11. Left Fallopian tube primary papillary adenocarcinoma. 25 ×.

down in hollow of sacrum. Left tube distended, about three to four times its normal size. Fimbriated ends of both tubes closed, right tube and ovary being incorporated in one mass. Left ovary grossly normal and left in situ. The left tubovarian mass and right distended tube were excised." The diagnosis of bilateral carcinoma of the Fallopian tubes was made by the pathologist.

The report of the macroscopic examination was as follows: "The specimen consists of both tubes and the right ovary. The right tube and ovary together measure 9 by 3.5 by 2.5 cm. They are intimately bound by firm dense adhesions. The tube has a boggy consistency and on cross section is found to be filled with a pinkish, gray, succulent tissue presenting a brain tissue consistency. The fimbriated end of the tube cannot be found. The ovary is completely replaced by a sac of corrugated wall. The cyst wall measures up to 0.5 cm. in thickness. The left tube measures 6 by 2.5 by 2 cm. It is tortuous, boggy in consistency, and the fimbriated end is obliterated. The distal third of the lumen of this tube is filled with tissue similar to that in the right tube."

The microscopic examination was as follows: "Both tubes are almost completely filled with cuboidal to columnar epithelial cells arranged in placques, strands, papillary processes and acini. The nuclei show marked variation, and numerous mitotic figures. The tubal walls are invaded." *Diagnosis*: Bilateral tubal adenocarcinoma.

A month later diagnostic curettage revealed a uterus three and one-half inches deep with no malignancy. The following month she was referred to the Brooklyn Cancer Institute for x-ray therapy. Examination, at this time, revealed a uterus slightly enlarged and fixed in position. On its left side, there was an adherent mass three inches in diameter. A total hysterectomy and left oophorectomy were performed. Metastatic carcinoma was found in the ovary but none in the uterus.

The macroscopic examination revealed the following: The specimen consisted of uterus, with its cervix, and a separate specimen which grossly appeared to contain tubal tissue. The uterus measured about 12.5 by 7.5 by 4.5 cm. Its serosa was granular and in certain areas hemorrhagic. Its adnexae were absent. On section the uterine wall appeared to be slightly thicker than normal, and grossly appeared to contain some fibrous strands. The endometrium was slightly congested and smooth. At one cornua, a very small, pin-head sized hemorrhagic cyst was found. The cervix was moderately hypertrophied, firm, and on section disclosed several small cysts containing mucus. The small separate mass described as appearing to be tubal tissue on section appeared to have a convoluted arrangement with some thickening of what appeared to be wall of the tube. The serosal parts of the tube appeared to be adherent to each other and the overlying mucosa was granular and hemorrhagic in appearance.

Microscopic examination showed that implanted on the surface of the ovary and invading its fibrocullular stroma were masses of

tumor cells arranged as multiple small papillae. Large areas were necrotic. The individual cells were columnar or polyhedral, having indefin-

FIG. 3. Case III. Bilateral Fallopian tubes primary adeno-carcinoma. 25 ×.

able cell boundaries, a pale pink cytoplasm, and varying sized, shaped, and stained vesicular or hyperchromatic nuclei which had prominent single or multiple nucleoli. There were scattered, regular mitotic figures. No tumor was present in the myometrium. The cervical lining was stratified squamous. There was a moderately heavy, subepithelial, lymphocytic infiltrate. The cervical glands were widely dilated. No tumor was present. *Diagnosis:* Metastatic papillary adenocarcinoma to left ovary.

The immediate postoperative course was uneventful. This was followed by the usual cycle of Roentgen therapy. Eleven months later she again came under our care at which time the entire pelvis was found to be filled with a large mass extending almost to the umbilicus compressing the rectum posteriorly against the sacrum and causing symptoms of partial intestinal obstruction. Proctoscopic examination showed a normal mucous membrane and the obstructive symptoms caused by extrinsic pressure. She complained of severe pelvic pain probably because of pressure on the sacral nerves. There was also a metastatic nodule in the posterior vaginal wall. This was radiated by a vaginal radium applicator containing three capsules for a total dosage of 1200 mgh. The patient became progressively worse and died within a month, which was just over a year from her initial admission to a hospital. Necropsy permission was refused.

CASE IV. P. P., a forty-five-year old, white,

married female was admitted to the hospital on December 16, 1940, with a history of vaginal spotting, bleeding, leukorrhea, and abdominal pain of one year's duration. Her menstrual and obstetrical histories were irrelevant. She had no weight loss or pain. She complained of moderate weakness. At thirty-two she had had an acute attack of salpingitis which subsided under conservative therapy. The preoperative findings were a large, palpable, abdominal mass of four months' duration. A supracervical hysterectomy and bilateral salpingo-oophorectomy were performed. The findings at operation were: "Uterus slightly enlarged and one small fibroid felt intramurally on the anterior surface. The abdomen was filled by two masses consisting of solid and cystic areas of a convoluted pattern and of a bluish discoloration, apparently, tubes and ovaries. The left ovary was discrete from the left tubal mass. The right tubo-ovarian mass extended to beneath the liver and was adherent to omentum and intestine by a few fine adhesions. The left tubal mass extended deep into the sacrum and was adherent with many fine bands. Each mass was roughly the size of two large male fists. No evidence of any peritoneal implantations." Five days later there was a disruption of the abdominal wound which required resuturing.

The macroscopic examination was reported as follows: "The uterus is small, irregular in outline due to two myomata and presents a polyp on the upper, anterior endometrium. Both tubes are tremendously elongated, distended, and twisted upon themselves. Both

ovaries are small, the left bound by dense adhesions to the tube. At the isthmic end of the left tube, a small bulbous enlargement is

Vaginal examination showed a parous introitus, cervical stump high in the vaginal vault pressed forward by an irregular mass filling the

Fig. 4. Case iv. Bilateral Fallopian tubes primary papillary adenocarcinoma. 40 ×.

noted. This presents a firmer consistency than the boggy consistency noted in the remainder of the tube. Both tubes contain serohemorrhagic fluid, but in the main are filled with papillary excrescences, both discrete and confluent, especially, on the left side, measuring up to 7 cm. in diameter. Both tubes are somewhat constricted near the cornual ends. The bulbous enlargement at the cornual end of the left tube is found to be due to complete filling of the lumen by the fragile, pearly-gray, friable tissue."

The microscopic examination revealed the following: "The tubal lumina are dilated and filled with masses of tumor cells arranged as multiple, papillary projections around narrow vascular cores. The cells are cuboidal to columnar and have varying sized, shaped and stained nuclei which are frequently undergoing mitotic division. There is widespread invasion of the tubal walls and myometrium by tumor cells which commonly are found in distinct lymphatic spaces. There is, also, invasion of the right tubo-ovarian ligament. The endometrial polyp is typical and benign.

Diagnosis: "Bilateral papillary adenocarcinoma of the tubes with extension to the uterus and ovaries. Fibromyomata of the uterus. Endometrial polyp."

One month after operation, she was referred to the Brooklyn Cancer Institute for Roentgen therapy. Her chief complaints were marked weakness and the loss of twenty-five pounds.

cul-de-sac. In the left vaginal wall there was a submucosal, nodular, metastatic mass. Rectal examination confirmed the vaginal findings. The usual cycle of Roentgen therapy was given without incident except for a herpes zoster and stormy menopausal symptoms. Her general and pelvic condition became progressively worse and she was transferred for custodial care four months after admission to our institution. Two months later she died and necropsy permission was refused.

CASE v. E. K., a fifty-two-year old, white, virgin female was admitted to the Brooklyn Cancer Institute January 8, 1935. Her symptoms were postmenopausal bleeding and intermittent lower abdominal pain. There was a mass in the cul-de-sac, the size of an orange originating on the left side, which at laparotomy proved to be an anaplastic, cystic carcinoma of the left Fallopian tube. Both ends of the tube were closed. This case has been ably reported by Dr. Herman Charache.[31]

SUMMARY

The literature of primary tubal carcinoma is reviewed. Salient clinical aspects are presented. The clinical triad of intermittent lower abdominal pain, relief by leucorrhea, and a receding palpable adnexal mass is emphasized. Unless this clinical entity is included in the differential, it is almost invariably overlooked in the

final diagnosis. Therapy to be rational must be based upon accurate clinical diagnosis, therefore, hormonal, pharmacological, x-ray, or radium therapy should not be administered symptomatically. During operation all specimens should be incised and examined macroscopically to determine whether more radical procedures are indicated. The only five cases that have occurred at the Brooklyn Cancer Institute are reviewed in this paper. Interestingly enough, they have all appeared during the last six years.

CONCLUSION

Primary tubal carcinoma is insidious in onset, evasive in clinical diagnosis and extremely rare. Our threshold of suspicion to this clinical entity must be lowered if we are not to overlook this diagnostic possibility, even though it is so uncommon. Prognosis is invariably fatal, therefore, early diagnosis and immediate radical therapy must be instituted.

REFERENCES

1. ORTHMANN, E. G. Centralbl f. Gynäk., p. 816, 1886.
2. ORTHMANN, E. G. Ztschr. f. Geburtsh. u. Gynäk., 53: 119, 1904.
3. ORTHMANN, E. G. Ztschr. f. Geburtsh. u. Gynäk., 58: 376, 1906.
4. FROMME und HEYNEMANN. Veits' Handbuch der Gynäkologie. Vol. 5, p. 164. J. F. Bergmann, 1910.
5. NÜRNBERGER, L. Veit-Stoeckel's Handbuch der Gynäkologie. 3rd ed., vol. VII, p. 729.
6. VON FRANQUÉ, O. Ztschr. f. Geburtsh u. Gynäk., 69: 409, 1911.
7. FRANK, ROBT. T. Gynecological and Obstetrical Pathology. P. 348. New York, 1922. D. Appleton & Co.
8. LYNCH, F. W. Pelvic Neoplasms. P. 394. New York, 1922. D. Appleton Co.
9. CALLAHAN, W. P. et al. Surg., Gynec. & Obst., 48: 14, 1929.
10. MEIGS, JOE VINCENT. Tumors of the Female Pelvic Organs. P. 364. New York, 1934. MacMillan Co.
11. ROBINSON, M. R. Am. J. Obst. & Gynec., 32: 84, 1936.
12. BARROWS, D. N. Am. J. Obst. & Gynec., 13: 710, 1927.
13. PHANEUF, L. E. Am. J. Surg., 39: 620, 1938.
14. HOLLAND, W. W. Surg., Gynec. & Obst., 51: 683, 1930.
15. HENDERSON, D. NELSON. Am. J. Obst. & Gynec., 37: 321, 1939.
16. WINTER, WERNER. Arch. f. Gynäk., 154: 374, 1933.
17. MARTZLOFF, KARL H. Am. J. Obst. & Gynec., 40: 804, 1940.
18. BOYD, WM. Surgical Pathology. P. 504. Philadelphia, 1933. W. B. Saunders.
19. VEST, CECIL W. A clinical study of primary cancer of the fallopian tube. Johns Hopkins Hosp. Bull., 25: 305, 1914.
20. VEST, CECIL W. Malignant Growths in Fallopian Tube. Practice of Surgery. Dean Lewis. Vol. 11, p. 25. W. F. Prior & Co. Loose-leaf system.
21. WILLIS, RUPERT A. The Spread of Tumours in the Human Body. P. 283. London, 1934. J. & A. Churchill.
22. DORAN, ALBAN. J. Obst. & Gynec., Brit. Empire, 17: 1, 1910.
23. WHARTON, L. R. and KROCK, F. H. Arch. Surg., 19: 848, 1929.
24. WECHSLER, H. F. Arch. Path., 2: 161, 1926.
25. WATKINS, R. E. and WILSON, W. M. Surg., Gynec. & Obst., 51: 1, 1930.
26. WATKINS, RAYMOND E. Treatment of Cancer & Allied Diseases. Pack & Livingston. Vol. 2, p. 1749. New York, 1940. Paul B. Hoeber.
27. GRAVES, WM. P. Gynecology. P. 534. Philadelphia, 1928. Saunders.
28. PARSONS, LANGDON. New England J. Med., 221: 367, 1939.
29. KEHRER. Monatschr. f. Geburtsh. u. Gynäk., 27: 327, 1908.
30. MATHEY, CORNAT, R. Radiotherapie Gynecologique. P. 274. Paris, 1936. Masson & Cie.
31. CHARACHE, H. Ann. Surg., 103: 290, 1936.

ACUTE APPENDICITIS IN INFANCY

ADOLPH H. EMERSON, M.D.

Assistant Attending in Pediatrics, Long Island College Hospital

BROOKLYN, NEW YORK

ACUTE appendicitis in infancy is a rare condition. Furthermore, it presents a difficult diagnostic problem. It was therefore thought advisable to record an additional instance of this disorder and point out the diagnostic difficulties encountered in appendicitis of very young infants.

An examination of the literature disclosed but a single paper which included a large series of reported cases of acute appendicitis in infants. This was a publication by Abt,[1] who listed eighty infants with appendicitis in his review of the literature from 1847 to 1917. Of these eighty infants thirty-seven were one year of age or younger.

Studies by other authors have emphasized the infrequency of appendicitis in infants, particularly in the group of infants less than one year of age. For example Beekman[2] stated that of forty-three cases of this disease in children under five years the youngest was twenty months. In a report by Busch[3] on 635 cases of acute appendicitis the youngest patient was eighteen months. Of 592 children less than twelve years of age who had appendicitis Potts[4] cited only three in the one year old group. Allen[5] reported only one infant under one year of age with acute appendicitis in his series of 612 children with this disease. In still another report[6] of 258 children suffering from this malady not one was under two years of age. A study by Hudson[7] was concerned with 848 children with appendicitis only two of whom were one year of age or less. Studies by Farr[8] and Christopher[9] have given additional data which indicate the rarity of acute appendicitis in the period of infancy.

CASE REPORT

P. H., a previously healthy and well nourished female infant of three months was seen at her home because of fever, irritability, anorexia and frequent crying attacks. The illness had begun twenty-four hours previously. During this period the infant had vomited once and had had one normal bowel movement. When first seen the infant did not appear very ill. A physical examination revealed an acute pharyngitis. The ear drums, lungs and heart appeared to be normal. The abdomen was symmetrical, full and moved freely with respirations. Gentle palpation in all regions of the abdomen appeared to distress the infant. However, there appeared to be no maximal point of tenderness and no abdominal masses were palpated. A rectal examination added no positive information. The temperature was 102°F.

The abdominal findings were interpreted as being due possibly to primary peritonitis or acute appendicitis, and hospitalization was ordered.

The laboratory findings were as follows: red blood cells—4 million, hemoglobin—11 Gm. per 100 cc., white blood cells 21,500; differential count, 68 per cent polymorphonuclear neutrophiles, 30 lymphocytes and 2 monocytes. Urine analysis was negative. Cultures of the urine and blood were sterile. A vaginal smear showed no organisms resembling gonococci. The nose and throat cultures revealed the presence of Staphylococcus aureus, hemolytic streptococcus and pneumococcus.

After a careful physical examination a surgical consultant advised further observation. During the night the infant took her formula well and was reported to have slept between feedings. A re-examination then showed a definite spasm of the rectus muscle in the right lower quadrant. There was also noted a sense of fullness and a definite point of tenderness in that area.

Operation by Dr. Charles B. Jones was performed under ether anesthesia. A McBurney incision was used. The peritoneum was found to be thickened and the vascularity increased. A small abscess cavity measuring 3 cm. was entered and one dram of purulent material evacuated. The walls of the abscess were thin and appeared to have been recently formed. The appendix, which formed a portion of the medial wall of the abscess, was easily identified and removed in the usual manner with inversion of the stump. In the central portion of the appendix was a gangrenous area and a perforation. Three Gm. of sulfathiazole powder were sifted into the abscess cavity and about the neighboring structures. One small cigarette drain was placed in the abscess cavity. The peritoneum and the abdominal wall tissues were then closed in layers.

Convalescence was stormy for the first two days but subsequently there was gradual improvement and the temperature became normal after the sixth postoperative day. Treatment included continuous intravenous infusions of normal saline with dextrose, citrated blood and sodium sulfathiazole. The level of this drug in the blood at no time rose above 5 mg. per cent. Drainage from the operative wound ceased in ten days. A culture of the abscess identified the offending organism as Bacillus coli.

The infant is now six months of age and in excellent physical condition.

COMMENT

Two characteristics of appendicitis in infancy are its insidious onset and the rapidity of its progress to gangrene and perforation. Even if seen in the first twenty-four hours the young patient is likely to have peritoneal involvement. This is usually in the form of generalized peritonitis, although sometimes a localized abscess is seen. It is generally believed that the ability of infants to localize such an infection is limited partly because the omentum in these small patients does not play the protective rôle attributed to it in adults. Moreover, the walls of the juvenile appendix have been described as being very thin and inelastic, thus predisposing to early rupture.[10]

The diagnosis of acute appendicitis in infants presents difficulties not usually seen in older children or adults. Heyl,[11] in referring to the disease in infants stated "it is likely that most in the first year and many in the second recover or die without the real diagnosis being suspected." Peterson[12] is of the opinion that many instances of gastrointestinal upsets, indigestion and colic may be examples of unrecognized appendicitis.

Abt[1] has discussed the diagnostic features of appendicitis in infants rather fully. He found that although constipation was the rule in severe types of infantile appendicitis, diarrhea may occur among the milder types, or diarrhea and constipation may alternate. A complicating generalized peritonitis may cause paralytic ileus with symptoms of bowel obstruction. A high fever may be present and polymorphonuclear leucocytosis is nearly always found. Vomiting, abdominal pain, distention and tumor mass were features of some of the cases discussed in Abt's review. This author emphasized that tenderness at McBurney's point is of diagnostic importance but that it cannot always be elicited. He added that the appendix may be deep and that abdominal tenderness may be greater on the left side than on the right, or that it may be deflected upward. A rectal examination is of great importance in such cases. Abt also analyzed his series of collected cases by age groups of from birth to three months, three months to six months and from six to twelve months.

The writer has studied a number of other published reports of acute appendicitis which occurred in infants less than one year of age. These publications show that this disease may occur at any time during the first year of life.

Several papers described what appeared to be acute appendicitis in the prenatal and the newborn periods.[13,14,15,16] Descriptions conforming to the classical picture of the disease as seen in adults were included.[17,18]

Campbell,[19] Ham,[20] Pope,[21] Langston,[22] and Rivarola[23] cited examples of acute

appendicitis complicating strangulated inguinal hernia in infants under one year of age. Abt's review[1] listed ten examples of this type. Burger,[24] O'Neill[25] and Lilienthal[26] also described hernial appendicitis in young infants. Gundobin[27] investigated the length of the appendix in a number of infants and found that the length varied from 3.4 cm to 11.6 cm. In 60 per cent of the cases examined the appendix extended into the small pelvis. McCarthy[28] is of the opinion that a long appendix might be caught in a hernial sac and thus become incarcerated, inflamed and gangrenous. The clinical picture of hernial appendicitis resembles that of strangulated hernia. In the former, however, the mass is tender and the overlying skin is soon likely to become red and hot suggesting the presence of an abscess. Black[29] has recently described an unusual case of an infant fourteen days of age with hernial appendicitis in which the appendix was contained alone in the sac.

Intussusception may be closely simulated by acute appendicitis in young infants. Coleman[30] saw an infant girl of five months who had been periodically screaming and drawing up her legs for nine hours. Bright red blood and mucus were passed by rectum. She had vomited once. The temperature was 100.2°F. A rectal examination was negative. Abdominal examination under anesthesia revealed a small lump deep under the right costal margin. Operation revealed an acutely inflamed appendix and free fluid in the abdominal cavity. There was no intussusception present. The lump was later thought to be the right kidney which had a prominent fetal lobulation. John,[31] Somers,[32] Heekes[33] and Crosier[34] described similar instances in young infants in which a preoperative diagnosis of intussusception was made, and although this condition was simulated clinically, acute appendicitis was found at operation.

As stated above many infants with with acute appendicitis are first seen by a physician after generalized peritonitis has complicated the original lesion. Vomiting, fever, distention, abdominal tenderness and either diarrhea or bowel obstruction associated with paralytic ileus may then be seen. If long continued, the resultant loss of fluids and electrolytes leads to dehydration, acidosis, stupor and collapse. It is the writer's opinion, based in part on some personal observations, that after a few days of such an illness, abdominal distention previously present may disappear and abdominal tenderness may no longer be detected. Kempton's[35] patient, when first seen by him on the eighth day of illness had neither abdominal distention nor muscle spasm, and yet autopsy revealed a perforated appendix and generalized peritonitis. Such an infant may be indistinguishable from one suffering with gastroenteritis with dehydration acidosis Incidentally, the opinion is held by some that enteritis may predispose to an attack of appendicitis.

In considering the differential diagnosis of acute appendicitis in infancy intussusception, gastroenteritis and hernial appendicitis have been mentioned. Pneumonia, intestinal obstruction, pleurisy, ileopsoas abscess and typhoid fever (rarely) are frequently confused with appendicitis.[1]

Primary peritonitis may be very difficult to distinguish clinically from acute appendicitis, or from peritonitis secondary to a ruptured appendix. Ladd,[36] however, advocates an operation "of a limited sort" for suspected primary peritonitis. During the course of this operation the status of the appendix is determined, and if involved in the infection it is dealt with in the usual manner.

REFERENCES

1. ABT, I. A. Appendicitis in infancy. *Arch. Pediat.*, 34: 641, 1917.
2. BEEKMAN, F. Acute appendicitis in the infant. *Ann. Surg.*, 80: 911, 1924.
3. BUSCH, IRVING and SPIVACK, A. H. Observations on acute appendicitis. *Surg., Gynec. & Obst.*, 70: 241, 1940.
4. POTTS, W. J. Acute appendicitis in children. *Am. J. Dis. Child.*, 55: 511, 1938.
5. ALLEN, PHILIP D. Acute appendicitis in children. *J. A. M. A.*, 109: 121, 1937.

6   STONE, C. S. Acute appendicitis in children. *Arch. Surg.*, 30: 346, 1935.

7. HUDSON, H. W. and CHAMBERLAIN, J. W. Acute appendicitis in childhood. *J. Pediat.*, 15: 408, 1939.

8. FARR, C. E. and BRAKELEY, E. Appendicitis in children. *Surg. Clin. North America*, October, 1928.

9. CHRISTOPHER, FREDERICK. Appendicitis in children under 5 years. *Am. J. Dis. Child.*, 31: 525, 1926.

10. KROCK, FRED., BILLINGSLEY, C. B. and WEDDINGTON, R. E. Acute appendicitis in infancy. *J. Arkansas M. Soc.*, 32: 161, 1936.

11. HEYL, J. H. Acute appendicitis in infants and children under 5 years of age. *New York State J. Med.*, 36: 332, 1936.

12. PETERSON, E. W. Appendicitis in infancy and the younger group of children. *J. M. A. Alabama*, 5: 405, 1936.

13. CORCORAN, WM. J. Prenatal rupture of the appendix. *Am. J. Dis. Child.*, 39: 277, 1930.

14. JACKSON, W. F. A case of prenatal appendicitis. *Am. J. Med. Sc.*, 127: 710, 1904.

15. HILL, W. B. and MASON, C. C. Appendicitis in a newborn. *Am. J. Dis. Child.*, 29: 86, 1925.

16. CH'ENG, Y. H. and K'ANG, H. J. Acute appendicitis in a newborn. *Chinese Med. J.*, 52: 876, 1937.

17. WERTHMANN, H. Appendicitis beim Kleinkind. *Zentralbl. f. Chir.*, 58: 1943, 1931.

18. LIPSHUTZ, BENJ. Appendicitis in infancy. *Arch. Pediat.*, 48: 649, 1931.

19. CAMPBELL, DONALD D. Acute perforative appendicitis. *Brit. M. J.*, 1: 1102, 1938.

20. HAM, C. I. Appendicitis in an infant. *Brit. M. J.*, 1: 1006, 1927.

21. POPE, SAMUEL. Acute appendicitis in an infant. *Brit. M. J.*, 2: 307, 1927.

22. LANGSTON, H. J. Acute gangrenous appendicitis and strangulated hernia in a 5 weeks old baby. *Virginia M. Monthly*, 57: 466, 1930.

23. RIVAROLA, JOSE E. Appendicitis herniaria en un nino de 45 dias de edad. *Prensa méd. argent.*, 20: 1671, 1933.

24. BURGER, T. O. and TORBERT, H. C. The diagnosis of acute hernial appendicitis. *Am. J. Surg.*, 42: 429, 1938.

25. O'NEILL, T. Appendix strangulated in congenital hernial sac in an infant. *Brit. M. J.*, 2: 87, 1941.

26. LILIENTHAL, H. Strangulated hernia and gangrenous appendix in a premature infant. *J. Mt. Sinai Hosp.*, 5: 607, 1939.

27. GUNDOBIN, N. P. Die Besonderheiten des Kindesalters. Allgemeine Medizinische Verlaganstalt, Berlin, 1921, page 307.

28. McCARTHY, P. A. and MAGRATH, J. L. Acute appendicitis in infants. *Pennsylvania M. J.*, 41: 5, 1937.

29. BLACK, B. M. and WAUGH, J. M. Hernial appendicitis report of 2 cases. *Proc. Staff Meet., Mayo Clin.*, 17: 21, 1942.

30. COLEMAN, F. H. Acute appendicitis in an infant resembling intussusception. *Lancet*, 1: 1008, 1936.

31. JOHN, R. W. Acute perforative appendicitis in an infant of twenty days. *Lancet*, 2: 1097, 1936.

32. SOMERS, R. H. Acute suppurative appendicitis in an infant of 3 months. *Northwest Med.*, 27: 344, 1928.

33. HEEKES, J. W. Appendicitis in an infant. *Brit. M. J.*, 1: 571, 1924.

34. CROSIER, M. Appendicite aigue chez un infant de huit mois. *Paris chir.*, 18: 297, 1926.

35. KEMPTON, R. M. Appendicitis under 2 years. *J. Michigan M. Soc.*, 31: 188, 1932.

36. LADD, W. E. and GROSS, R. E. Abdominal Surgery of Infancy and Childhood. P. 192. Philadelphia, 1941. W. B. Saunders Co.

COMPLEX TRIDERMAL TERATOMA OF THE STOMACH (BENIGN)

CASE REPORT

ALEXANDER N. SELMAN, M.D.

Attending Surgeon, Nyack Hospital, Nyack, New York, and Rockland State Hospital, Orangeburg, New York

SPRING VALLEY, NEW YORK

TRIDERMAL teratoma of the stomach is a very rare condition and extensive search of the literature fails to record such a case. Boyd,[1] Ewing[2] and Ballantine[3] fail to report a single instance. Eustermann and Balfour,[4] discussing non-malignant tumors of the stomach found at the Mayo Clinic, report twenty-six benign tumors, none of which was a teratoma. Lexer,[5] Askanasy,[6] Hosmer,[7] Marchand,[8] Ahrens,[9] and Englander[10] have reported cases of tridermal teratomas of the peritoneum but these tumors were either situated behind the liver or located in the transverse mesocolon. Some of the latter were difficult to distinguish from retroperitoneal tumors. Brouha,[11] and Schonholzer[12] describe several unattached tridermal teratomas occurring retroperitoneally. Borst[13] and Bolzana[14] describe several cases derived from the ovary and also retroperitoneal. Polony[15] described a case of exogastric teratoma occurring in a six year old child. Covered by the gastrocolic ligament, the tumor was attached to the stomach by a pedicle. It consisted of numerous cysts containing a stringy fluid and gelatinous, purulent or caseous masses. Many of the cysts were secondarily infected. Histologically, the cysts were closed and many were lined with epithelium of a digestive type but without glands. The stroma and intracystic septa were composed of connective tissue and muscle fibers. Differentiating it from a Schwannoma by the absence of neuro tissue he made the diagnosis of simple teratoma with secondary infection.

Teratomas of other organs such as thyroid, brain, testicle, ovary, kidney and others, are very common but the absence in the literature of a case of complex tridermal teratoma of the stomach (benign) and the fact that it was successfully removed from a child four months of age, is the purpose of this report.

CASE REPORT

E. B., a male, white child, was born at term May 30, 1941. The delivery was normal, birth weight seven pounds, seven ounces. Circumcision was done on the eighth day and the wound healed rapidly without incident. The child appeared normal except for a hemangioma one inch in diameter present in the midline, about two and one-half inches above the umbilicus. Routine examination at the age of six weeks revealed no abnormalities. The weight at this time was eight pounds, twelve ounces. The hemangioma apparently had not grown any in size. The liver and spleen were palpable, otherwise the abdomen felt normal. The child was taking its formula well, the bowels were moving satisfactorily and the stools appeared well formed and brown in color.

Routine examination on August 28, 1941, at the age of three months, revealed a large mass occupying the whole upper left quadrant of the abdomen extending down below the umbilicus. The mass was freely movable and not adherent to the skin and apparently not tender to touch. It could be shifted up beneath the ribs with ease. The liver was palpable but not so the spleen. The hemangioma had not noticeably changed in size. The child weighed eleven pounds and six ounces, was taking its feedings well, the bowels were moving well and there was no blood in the stools or urine.

The child was admitted to Nyack Hospital for x-ray studies. Flat plates of the abdomen were taken on August 29, 1941. The roentgenologist reported, "There is a rather large, roughly, spherical mass in the left side of the abdomen occupying most of the upper left

quadrant and extending down below the umbilicus. It seems cystic in consistency but the origin is not clear. It does not seem that

lieved to be the kidneys did not correspond to the palpable mass." The child was examined by numerous members of the surgical staff at
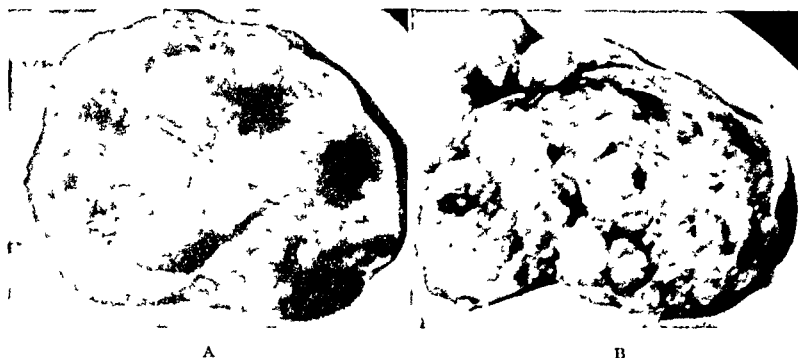
FIG. 1. A, gross appearance of the tumor, 15 by 10 by 8 cm. B, the tumor sectioned in half.

FIG. 2. Microphotograph showing area of skin with keratin layer, and true derma containing sebaceous glands, hair follicles and fatty tissue.

the spleen is displaced." On September 4, 1941 blood count revealed—hemoglobin Sahli 70 per cent (14.0 Gm.), red blood count 3,800,000, white blood count 8,000, neutrophils 32 per cent, lymphocytes 56 per cent, mononuclear leucocytes 10 per cent, eosinophils 2 per cent, blood smear showed some hypochromia. The Wassermann reaction was negative. On September 5, 1941, an intravenous pyelogram was done. The roentgenologist reported, "The large amount of stool and gas in the intestines and gastric distention with gas make clear visualization difficult. The dye is apparently excreted well as shadows of the bladder are obtained very promptly. The calyces and pelves of the kidneys are not seen. The outline be-

the hospital and these concurred in a diagnosis of probable Wilms tumor of the left kidney, although they were not able to rule out intraperitoneal lesion completely. Urological consultation agreed with the probable diagnosis of a kidney lesion. Arrangements were made for operative intervention, and this was done on October 3, 1941.

Drop ether was used for anesthesia. Because of the doubt as to the exact lesion, I chose an incision about one inch lateral to the outer border of the rectus intending to add a T if a kidney lesion was found. On approaching the peritoneum it became obvious that the lesion was intraperitoneal. The peritoneum was incised in the line of the skin incision and the

abdomen explored. A fibrocystic mass almost six inches in diameter was found in the upper portion of the abdomen. It was delivered through the incision. Further examination found that it was growing from the stomach. No palpable glands were noted about the stomach and there was no involvement of the liver. The mass was not adherent to the adjacent organs and was attached to the stomach anteriorly on the greater curvature about half way between the cardia and pylorus by a broad base. Clamps were applied to the stomach beyond the base of the mass and the entire mass removed *in toto*. Inspection of the stomach inside and without showed no other involvement. The large rent in the stomach was closed with one row of through-and-through interlocking sutures covered over by two rows of continuous seromuscular Lembert sutures. The abdomen was closed in layers without drainage. Three silk through-and-through sutures were used as retentions. The convalescence was uneventful.

Clyses of 150 cc. 3 per cent glucose in saline were given every four hours for the first twenty-four hours, every six hours for the second twenty-four hours and twice during the third twenty-four hours. Dextrimaltose in water, by mouth, was started twenty-four hours postoperatively and continued every two hours for twenty-four hours. A milk formula was commenced after forty-eight hours and orange juice and cereal were added on the sixth day. Sutures were removed on the ninth day and the

The child has continued to thrive and develop normally since leaving the hospital and when last examined on December 1, 1941, was
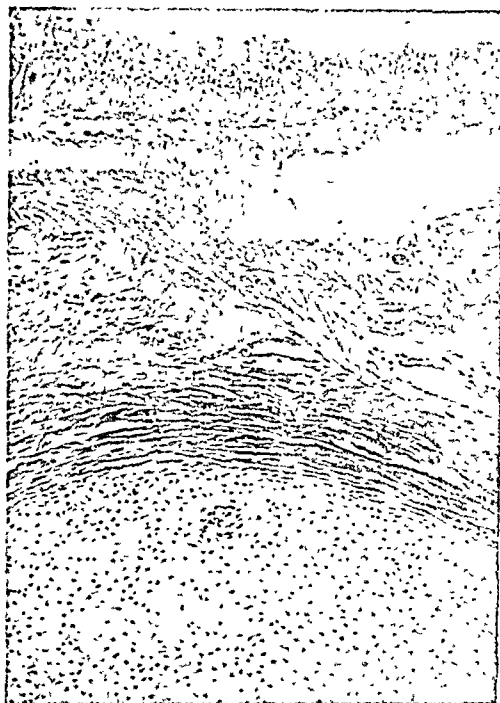
Fig. 3. Microphotograph showing bronchus with cartilage, smooth muscle and mucosa.

found to weigh fourteen pounds six ounces. The wound has healed solidly and there have been no digestive or other disturbances of any kind.

Pathological Report by Dr. W. R. Strutton.
Gross Description: The tumor was a bluish
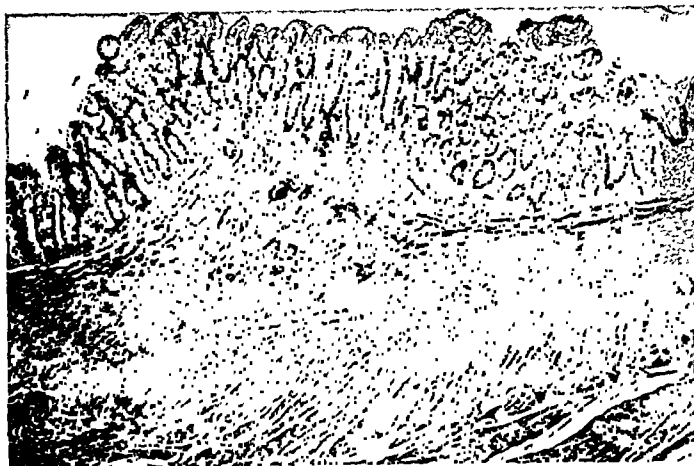
Fig. 4. Microphotograph showing intestinal structures found—colon.

child was discharged on the thirteenth day postoperatively. The wound healed and the child was taking its regular feedings.

white, rounded ovoid mass 15 by 10 by 8 cm. covered by a fibrous-like tissue with cystic areas showing beneath the capsule. On section

it showed numerous cysts, tough fibrous areas, cartilage and some hemorrhagic areas. On palpation it showed numerous areas irregularly hardened, soft and tough to touch, with cysts extending through the cut surface, interspersed with hardened cartilagenous, whitish areas, tough fibrous interlacing partitions giving a variegated appearance to the cut surface. The tumor was attached to the stomach wall by a base 5 cm. in diameter. This base was intimately adherent through the serosa and muscularis and covered only by gastric mucosa.

Microscopically, sections showed a multiplicity of tissues, the best defined being skin with hair follicles, sweat glands, fibrous, fatty and myxomatous tissues. Other areas showed smooth muscle, blood vessels, serous cysts lined by flattened epithelium, a deeply pigmented surface of cartilage, poorly defined, but suggesting bronchi, mucous membranes lined by tall columnar epithelium containing mucus, tubular glands and other areas of transitional epithelium. The tubular glands resembled colon and stomach tissues.

Diagnosis: Complex teratoma (benign).

The tumor was also viewed by Dr. Fred Stewart of the Memorial Hospital, New York City, who took sections and made slides. He concurred in the diagnosis, calling it a complex tridermal teratoma—benign. Dr. Sidney Farber of the Children's Hospital in Boston, viewed the slides and also concurred in the diagnosis.

REFERENCES

1. BOYD, WM. Surgical Pathology. Chap. 15, page 290. Philadelphia, 1939. Saunders.
2. EWING, JAMES. Neoplastic Diseases. Chap. 49. Philadelphia, 1934. Saunders.
3. BALLANTYNE. Ante-natal Pathology.
4. EUSTERMANN and BALFOUR. The Stomach and Duodenum. Page 547. Philadelphia, 1936. Saunders.
5. LEXER. *Langerbeck's Arch. f. Chir.,* 61: 648, 1900.
6. ASKANASY. *Deutsche path. Gesellsch.,* 8, 11: 39, 1907.
7. HOSMER. *Boston M. & S. J.,* 102: 61, 1880.
8. MARCHAND. *Breslau artzl. Zeitung,* 3: 237, 1881.
9. AHRENS. *Langerbeck's Arch. f. Chir.,* 64: 426, 1901.
10. ENGLANDER. *Centralbl. f. allg. Path. u. path. Anat.,* 13: 393, 1902.
11. BROUHA. *Rev. de gynec. e d'obst.,* 5: 231, 1902.
12. SCHONHOLZER. *Ziegler's Beitr. z. path. Anat.,* 40: 349, 1907.
13. BORST. *Centralbl. f. allg. Path. u. path. Anat.,* 9: 449, 1898.
14. BOLZANA. (1. D. Wurtzburg 1901.) (From Ewing.[2])
15. POLONY. (de Befort.) *Mém. Acad. de chir.,* 62: 622, 1936.

TORSION AND GANGRENE OF HYDATID OF MORGAGNI [*]

REVIEW OF LITERATURE AND CASE REPORT

JOHN W. HOWSER, M.D.   AND   LOUIS P. RIVER, M.D.

Associate Attending Surgeon, Cook County Hospital   Attending Surgeon, Cook County Hospital

OAK PARK, ILLINOIS

VESTIGES of anlage of the genito-urinary systems remain in both sexes in the adult. Most of them are rudimentary, small structures and cause no pathologic manifestations in adult life. One of these structures was described by Morgagni in the seventeenth century and has been known since as the Hydatid of Morgagni. Other correct terms are the vesicular appendage or appendix vesiculosa of the Fallopian tube, or testis. It is peculiar in that it represents an embryonic vestige, which is not utilized in the adult in either sex. Because of this, it remains a relatively similar structure, except in position, in both the male and female. These hydatids are thought to be remnants of the segmented cephalic mesonephric tubules which do not enter into the formation of either the epoophoron or the paroophoron.[1,2] Arey[3] believes that they may also represent a cystic rudimentary Muellerian funnel accessory, formed when the Muellerian duct begins to roof over the cleft from which it is formed.

In the female adult, these structures appear as ovoid to spherical hydatids, usually with a small pedicle, hanging from the broad ligament, just below the fimbria of the tube. They usually are anteriorly placed.[4] They may appear as two or three fused vesicles with constrictions between each segment.

In the male adult, these hydatids, known as the appendix of the testis, appear as minute oval, sessile bodies, attached to the upper extremity of the testis,[4] just beneath the head of the epididymis.

The size of these hydatids varies, but they are usually about .9 cm by 1.0 cm.

They may be sessile, but ordinarily they have a pedicle, which may reach one inch in length. The length of the pedicle undoubtedly plays a part in the torsion of such a hydatid.

Apparently, the pedunculated hydatid is relatively common, as many are seen during routine pelvic explorations.[32] In this case, torsion of hydatids, with acute symptoms, might be common. However, this is not so. In reviewing the literature we found reports of only thirty-nine cases. Of these, twenty-six, or 66.67 per cent have been in male patients. The remainder, thirteen, or 33.3 per cent have been in females. The higher incidence in males is thought to be the result of the motility of the testis, hanging free in the scrotum. Add to this, a hydatid, itself with a pedicle, and it is easy to see how torsion can occur. The usual manifestations of torsion in the male, are similar to those of torsion of the testis itself; in a high percentage of cases this has been the preoperative diagnosis. However, Mouchet[8] saw four cases in which there was a "silent" torsion, with symptoms over a considerable period. Also, it is possible to have the development of a hydrocele, as reported by Contiades[13] with the diagnosis of a traumatic orchitis.

In the male, with bilaterally descended testicles, the usual findings are in the scrotum; hence, there is little confusion between the symptoms of torsion of testis, cord or testicular appendage, and those of acute abdominal disease.

In the male with ectopic testicles, torsion of a hydatid of Morgagni will cause symptoms dependent upon the position

of the ectopic organ. That this may and does occur is evidenced by the cases reported by Monclav[14] and also by Chidi-
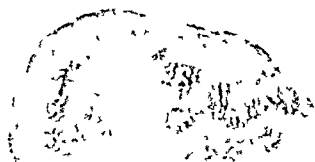
FIG. 1. (Cook County Hospital No. 41-68813). Photograph of a hemorrhagic, gangrenous hydatid of Morgagni found in a white female, fourteen years of age.

chimo.[34] In the latter's case the patient had a strangulated hydatid, with a cryptorchid and congenital hernia. The findings were those of the acute surgical abdominal catastrophe. The remaining reports of cases in males record no features at variance with those of Bonnet[7] and others.[8—13,15,17—18,20—21,23—24,26—27,29—31,31—36]

It is in the female, in whom the hydatid of Morgagni is normally found in the abdomen, that one may expect to find torsion representing the picture of acute abdominal disease. The preoperative diagnosis is seldom if ever made, the most usual impression being that of either acute appendicitis or twisted ovarian cyst. Waugh[32] points out that the reports in the literature indicate that more of these hydatids with torsion are found on the right side. He accounts for this fact with the observation that right sided abdominal pain is viewed as more serious and hence laparotomy is advised, whereas left sided abdominal pain is usually conservatively treated. There are no reports in the literature of torsion, strangulation and gangrene of a hydatid of Morgagni causing death. This may be accounted for by the fact that the hydatid is small, and does not communicate either with a fecal containing viscus or any functioning organ.

Waugh[32] believes that the frequency of this picture of torsion must be greater than reports indicate, and that many must have been overlooked when a benign appendix was removed. The succeeding clinical picture occurring as a result of the gangrene of the hydatid could be misinterpreted as "gas pains" or some other such complications of the appendectomy.

The first report of torsion of a Morgagni hydatid found as a cause of acute abdominal disease was made by Andrews[5] in 1912. The patient was six months pregnant. Apart from this complication, the subsequently reported cases vary little.[6,16,19,22,25,28,32,37]

The most frequently encountered clinical findings are similar to, often less severe than those of an ovarian cyst with twisted pedicle. The onset is usually acute, of varied severity, with from moderate pain to extreme prostration. This initial pain is soon replaced by crampy, colicky pain, with increasing severity, and shorter intervals which are pain free, until as torsion is completely established and hemorrhagic infarction occurs, the pain is steady and dull. The physical findings are moderate to severe tenderness in the lower quadrant of the affected side, somewhat lower than one might expect in acute appendicitis. On pelvic examination, there may be extreme tenderness upon manipulation of the adnexa of the affected side. Frequently, however, the patient has been a young girl and pelvic examination has been hampered by an intact hymen.

Because of the infrequent occurrence of torsion of hydatids of Morgagni in the female, the following case is reported. It is the fourteenth recorded case.

CASE REPORT

M. S. (No. 41-68813), a white female of fourteen years of age, was admitted to the Cook County Hospital on December 10, 1941. Her complaints upon admission were cramp-like, colicky abdominal pains beginning nineteen hours previously. She had menstruated from December 4 to 8, a period normal in every

respect. The onset of the colicky pain was in the right lower quadrant, severe, causing her to stay in bed from the onset. After the first pain, which stayed with her steadily for one hour, the character of the pain changed. Instead of the steady pain, there occurred wave-like pains, lasting from one to three minutes, which would then disappear, leaving her fairly comfortable in the interim. At first these pains were about thirty minutes apart, but the interval between pains was getting shorter, so that on admission they were occurring almost continually. About 5 P.M., ten hours after onset, she vomited food previously taken, which was a glass of milk taken at 3 P.M. At 12:30 P.M. she had a normal bowel movement which had no effect on the pain.

The inventory by systems was essentially negative. The menses had begun at eleven years of age; there were twenty-eight day intervals, with a moderate four-day flow.

Physical examination revealed a well developed, well nourished white female of fourteen, acutely ill. Her temperature was 99.8°F. rectally; pulse 68–74, blood pressure 116/75.

Head, neck and chest were negative except for slight hyperemia of the pharynx. The abdomen was scaphoid; liver, kidney and spleen were not palpated. No masses were palpable. There was moderate tenderness over McBurney's point on deep palpation but no rebound tenderness. Psoas, obturator and Rovsings signs were negative. Rectal examination revealed nothing abnormal. The external genitalia were normal and the hymen intact.

Laboratory workup was as follows: Vaginal, urethral smear negative for gonococcus; urinalysis negative; white cell count 16,500.

A tentative diagnosis of ruptured corpus hemorrhagicum was made, and conservative treatment instituted, with frequent re-observations. It was felt that the repeated colicky type of pain did not indicate appendicitis and that a pelvic lesion was more likely.

The patient spent an uncomfortable night, the colic recurring, perhaps somewhat less intense the first part of the night. Eight hours later, or about twenty-four hours after onset, the white blood count was 18,500. There was definite right lower quadrant rigidity, tenderness and rebound tenderness. A probable diagnosis of acute appendicitis was made and laparotomy performed.

Under ether, nitrous oxide and oxygen anesthesia a McBurney incision was made. A normal appendix was easily delivered. The terminal 25 cm. of ileum was examined and found to be normal. It was then noted that there was a small amount of sanguineous fluid escaping from the right side of the pelvis. The McBurney incision was extended into the rectus sheath, as advocated by Weir, and the right adnexa delivered. Observed on the anterior surface of the right broad ligament, just below the fimbria, was a twisted, hemorrhagic Morgagni hydatid of 10 by 8 mm. The remainder of the right adnexa was normal. The hydatid was removed by ligature and excision of the base; the tube and ovary were left undisturbed. Hemostasis was effected with suture. Peritonization of the raw surface of the stalk of the hydatid was effected. The appendix was removed and the stump inverted. The peritoneum was closed with catgut, the fascia, muscles and skin with interrupted steel wire .010 mm. in diameter. The patient made an uneventful recovery and left the hospital on the sixth postoperative day. She has been followed for four months, has a normal menstrual history, and no further right lower quadrant pain.

It would seem to be good judgment during exploration of the right lower quadrant for a suspected acute appendicitis, to investigate the adnexa for such strangulated hydatids, in the event that the appendix is not the cause of the trouble.

In the event of torsion and strangulation of a hydatid of Morgagni, the treatment of choice is ligation and excision, with peritonealization of the base.

SUMMARY

1. A brief embryologic discussion is presented as to the origin of hydatids of Morgagni.

2. A review of the literature has shown that torsion of such hydatids has been reported in thirty-nine instances.

3. Torsion of hydatids of Morgagni occur more commonly in the male, the ratio being two to one.

4. A brief discussion of the symptomatology and physical findings is presented.

5. A case of torsion of a right hydatid of Morgagni in a girl fourteen years of age, treated by ligation and excision, together with an appendectomy is reported.

6. It is suggested that in all cases of right lower quadrant pain, if a normal appendix is found at operation, the right adnexa be inspected for torsion of such hydatids.

REFERENCES

1. AREY, L. B. Developmental Anatomy. 4th ed. Philadelphia, 1940. W. B. Saunders.
2. CULLEN, THOMAS. Johns Hopkins Hosp. Bull., vol. 16, 1904.
3. AREY, L. B. Personal communication.
4. GRAY, HENRY. Anatomy of the Human Body. 22nd. ed. Philadelphia. Lea & Febiger.
5. ANDREWS, H. R. J. Obst. & Gynaec., Brit. Emp. 22; 210, 1912.
6. WATERS, C. H. J. A. M. A., 72: 1072, 1919.
7. BONNET, A. Arch. soc. d. sc. méd et biol. de Montpelier, 9: 183, 1928.
8. MOUCHET, A. Presse méd., 36: 1345, 1928.
9. BROSTER, L. R. Brit. M. J., 1: 145, 1929.
10. LAGOS, GARCIA. Semana méd., 2: 836, 1929.
11. DIX, V. W. Ztschr. f. urol. Chir., 33: 486, 1931.
12. FOSHEE, C. H. J. A. M. A., 99: 289, 1932.
13. CONTIADES, X. J. Ann. d'anat. Path., 9: 795, 1932.
14. MONCALV, L. J. d'urol., 35: 501, 1933.
15. GUILLERNIN. Rev. méd. de l'est, 61: 826, 1933.
16. MacDERMOTT, E. Brit. M. J., 2: 679, 1934.
17. ROCHE, A. Clin. J., 64: 960, 1935.
18. RHODES, J. S. New England M. J., 213: 1005, 1935.
19. TRASTENBERG, J. Am. J. Obst. & Gynec., 31: 162, 1935.
20. TAVERNIER et al. Lyon chir., 33: 598, 1936.
21. GRASSO, R. Arch. ital di chir., 43: 221, 1936.
22. TAILHEFER, A. Mém. Acad. de chir., 62: 975, 1936.
23. CASTRO, H. D. Mém. Soc. urug. de urol., 2: 36, 1936.
24. OLASCOAGO, M. L. Semana méd., 2: 1484, 1936.
25. GREGOVOVA. Českoslor. gynaek., 16: 224, 1937.
26. LAMBERT, J. Brit. J. Surg., 25: 553, 1938.
27. GUERIN, R. J. de méd. de Bordeaux, 115: 71, 1938.
28. CETRONI, M. B. Ann. di ostet. e. ginec., 61: 387, 1939.
29. ROLNICK, H. C. J. Urol., 42: 458, 1939.
30. RANDALL, A. J. Urol., 41: 715, 1939.
31. McFADDEN, G. D. F. Lancet, 1: 320, 1939.
32. WAUGH, J. M. Proc. Staff Meet., Mayo Clin., 14: 23, 1939.
33. FORT, A. An. de cir., 5: 160, 1939.
34. CHIDICHIMO, G. Policlinico, 47: 1806, 1940.
35. SCOTT, R. T. J. Urol., 44: 735, 1940.
36. BRYAN, T. B. L. East African M. J., 18: 146, 1941.
37. BOWLES, H. E. Am. J. Obst. & Gynec., 35: 706–708, 1938.

LUXATION OF TESTES

CASE REPORT

PETER J. SHAMMON, M.D.

Assistant Attending Surgeon, Kings County and Caledonian Hospitals

BROOKLYN, NEW YORK

SPORADIC cases of traumatic dislocation of testicle are occasionally reported and the reader is always reminded of the rarity of such condition. The earliest case on record was reported by Claubry in 1818. Luxation may be of one or both testicles, may be simple or compound, but the latter is considered extremely rare. Only three cases were collected by Alyea in 1929 and in 1940 Miller and Mickley reported one case of compound dislocation of both testicles. C Lewis Gaulden, of Los Angeles, found only five cases of dislocation of both testicles in 1922 to which he added a case of his own. While in some of the cases reported the exact nature of the trauma producing luxation was not described, the great majority of dislocations were caused by direct trauma to the part or adjacent areas. Herbst and Polkey gave an excellent review of all cases reported up to 1934. They summarized some ninety-three cases collected; sixty-six were simple dislocations of one or rarely both testes, and twenty-seven were so-called "herniae testes." Of the sixty-six cases of simple dislocations, forty-three were due to direct trauma, three had no reported cause, eleven were caused by effort trauma and seven by mechanical prevention of descent of congenitally undescended testes, such as the use of truss for false diagnosis of hernia. That review did not include cases of self-mutilation by bandage, truss or ligature. Seven of such cases were reported and nearly all were in the Russian army. Of the twenty-seven herniae testes reviewed, two occurred during an operation on the cord, the rest by direct trauma. There were three cases of simple dislocation of both testicles, two of compound dislocation, four were

penile, six anal, one crural and one into the hip joint. All others were toward the inguinal region and into the canal.

Spontaneous reduction was accomplished in a few cases, but the majority were reduced by operation. In six cases castration was necessary. Two patients died from the injury, one by the nonreduced dislocation becoming cancerous, and the other from herniation shock. Dor reported a case of Donnet in 1937 in which luxation of both testicles occurred which was reduced surgically. McCrean, in 1938, reported a case of spontaneous reduction four days after the injury which caused the ectopia although gentle manipulation failed two days before. The case reported at this time is a case of luxation "par effort."

CASE REPORT

H. T. R., age forty-two, a white, American born, airline worker and veteran of World War I, with several subsequent enlistments in military service, was admitted to Caledonian Hospital complaining of a painful mass in the right groin. The following history was obtained:

On February 24, 1941, about 10 P.M., he was coming down a ladder carrying a propeller dome, which began to slide off his arms but was held as he came down to the ground with some force. He felt faint and experienced a tearing sensation in the right groin, but continued to work until the afternoon of February 27, 1941, when the pain became intense. A physician was then consulted and a diagnosis of strangulated hernia was made. The correct diagnosis of luxation of right testicle was arrived at upon admission when an empty right scrotal sac was discovered. The testicle was felt above Poupart's ligament and could not be reduced manually. An operation was performed on March 3, 1941, and a strangulated testicle was found resting on the aponeurosis of

the right external oblique muscle, above and lateral to the external inguinal ring. The organ was surrounded by particles of blood clots. The spermatic cord was twisted, contracted and enlarged to three times its normal size. The testicle was dark blue in color due to interference with its blood supply by the congested and strangulated cord as it emerged through the external ring. The ring was open and the cord dissected up into the pelvis but the blood supply remained impaired and the cord contracted. The removal of the testicle was deemed advisable. The cord was divided between two ligatures and the stump sutured to the border of the internal oblique muscle. The fascia of the external oblique muscle was repaired as in a herniorrhaphy. The postoperative course was uneventful.

Pathological Report. "Gross specimen consisted of a testicle the tunica of which is hemorrhagic on section. The upper pole shows hemorrhagic areas. Histology: Section of the testicle and epididymis shows marked edema, cell infiltration and hemorrhagic infarcts particularly in the epididymis."

SUMMARY

One case of traumatic dislocation of testicle is reported making a total of 100 cases reported to date, including the herniae testes.

REFERENCES

GAULDEN, C. LEWIS. *California State J. Med.*, vol. 20, 1922.

LEGUEU. L'orchite traumatique. *Monde méd.*, Paris, 32: 745, 1923.

HERBST and POLKEY. *Am. J. Surg.*, 34: 18, 1936.

HUDDY. *Brit. J. Urol.*, 1: 187, 1929.

DOR. *Bull. et mém. Soc. d chirurgiens de Marseille*, 11: 255, 1937.

MILLER and MICKLEY. *J. A. M. A.*, May 11, 1940.

MAUCLAIRE. Hernie traumatique. *Bull. et mém. Soc. d chirurgiens de Paris*, 11: 1263, 1914.

PRIMARY HODGKIN'S SARCOMA OF THE JEJUNUM WITH PERFORATION RESECTION AND RADIOTHERAPY

CASE REPORT

PASQUALE D. BADIA, M.D.

Assistant Surgeon, Morrisania City Hospital

NEW YORK, NEW YORK

THE infrequent occurrence of primary Hodgkin's disease of the small intestine and the singular circumstance which resulted in the perforation of a loop of the jejunum, associated with an extensive inflammatory process of the gut and the atypical clinical findings, are all interesting observations which make this case worthy of publication.

Hodgkin's disease is today a well recognized morbid entity characterized by specific histopathologic lesions of the lymph glands and the lymphoid tissue of the body, associated with an anemia, a varied symptomatology and a fatal course. In reading the literature, all the various synonyms for the disease may create considerable confusion as to classification and causation and yet the histologic picture and pathologic behavior has remained so contant for the past four decades as to avoid any possibility of confusion.

The disease is of relatively low frequency; it seems to occur twice as frequently in males as in females. Heredity, environment, occupation and food play no important rôle. It attacks all races and all nationalities alike. The reports of cases in America seem to indicate that the distribution is more among the whites and the American born (a study of 214 cases by Peirce, Jacox and Hildreth at Michigan).

It can occur at any age but the largest number of patients are observed during the second and third decade of life. Smith collected five cases in which the onset was before the sixth month of age. The youngest patient recorded in the literature is that of Priesel and Winkelbauer, a girl four and one-half months old who died of this disease which had been present since birth. A cervical node removed from the mother in the last month of pregnancy showed Hodgkin's disease.

Notwithstanding the extensive research work done since 1832 when Hodgkin's first series of cases were presented, the etiology of the disease still remains obscure. Opinions are conflicting as to causation. Some observers attribute it to various types of chronic infections including tuberculosis; the majority of investigators lean more toward the theory that it is of neoplastic origin. Sternberg (1898) maintained that the disease was closely related to tuberculosis, whereas Reed (1902) believed that the process is an infective granuloma of unknown cause. Both clarified the histologic picture of the disease by placing particular emphasis on the findings of multinuclear giant cells, the Sternberg or the Dorothy Reed giant cells.

In 1932, Gordon described a test which is a valuable confirmatory test when accompanied by positive histologic examination. He injected intracerebrally a broth suspension of diseased lymph-nodes into rabbits and guinea pigs. This was followed by a syndrome (bearing his name) of spastic paralysis, ataxia, retraction of the head, fits and loss of weight. The test was positive in 70 per cent of his cases. Gordon believed that the disease was caused by a virus. Steiner found the test positive in 73.9 per cent of 299 recorded cases of histologically proved Hodgkin's disease and falsely positive in only 1.77 per cent of 452 control cases. In 1938, several independent workers (D. S. Edward,

McNaught, J. B. Turner, Jankson, and Parker) demonstrated that the Gordon syndrome was caused by the eosinophils

viz., loss of weight, weakness, loss of appetite, pallor and myelopthisic anemia. Fever is present during the course of the

A   B
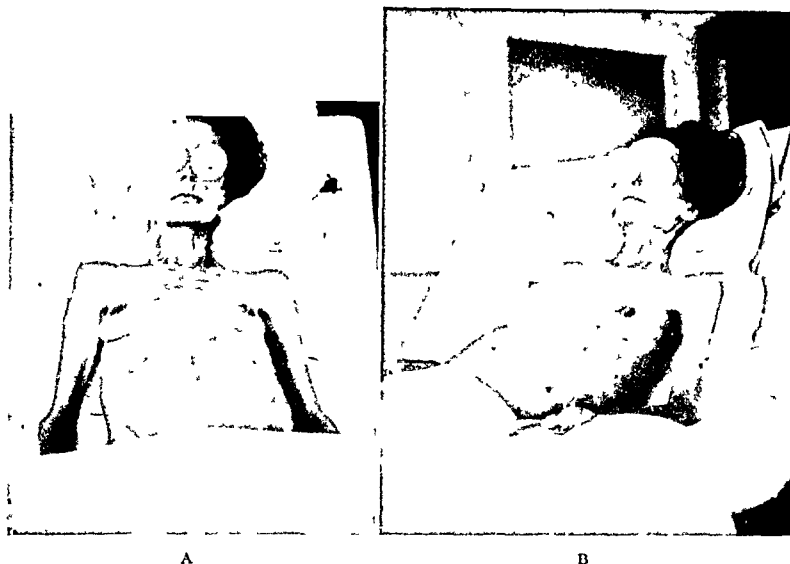
FIG. 1. A and B, showing patient with marked cachexia.

present in the tissues affected by Hodgkin's disease and that the same syndrome can be produced by other tissues containing eosinophils.

Epstein in a series of cases gathered from the literature emphasized the fact that the disease is not only less frequent in females, but that one-half or more women with Hodgkin's disease survive more than five years and that one fifth survive ten years. His findings are not in harmony with those of other observers.

Finzi, at the Fifth International Congress of Radiology in 1937, reported cases of patients who lived as long as eighteen years without evidence of active disease. Best results have been obtained in patients in whom the disease was confined to the cervical region and treated by heavy irradiation. Finzi believes that when the disease is localized it can be cured.

In a large percentage of cases the disease seems to begin with a painless enlargement of lymph-nodes in the left side of the neck. Many investigators have pointed out that this region is the most common initial involvement. As the disease progresses, certain constitutional symptoms appear,

disease in most all cases. Some cases show the Pel-Ebstein variety of pyrexia, (febrile periods persisting for two or three weeks, alternating with afebrile periods of a week or ten days' duration). In advanced cases there is marked elevation of pulse out of proportion to the temperature. Fever is usually a sign of deep structure involvement.

The blood picture does not show any special characteristics of the disease; 16 per cent show a leukocytosis and 5 per cent have eosinophilia. Anemia is always present in those patients with systemic symptoms. Some workers have demonstrated the co-existence of Brucella infection and Hodgkin's disease by isolating the Brucella melitensis from blood or lymph-nodes. These observations do not establish an etiological relationship to Hodgkin's disease and it suggested that the clinical course of Hodgkin's disease may be influenced by Brucellosis.

The following is the order of the sites of origin most commonly found: Left neck, right neck, both sides of neck, mediastinum, right axilla, left axilla, left groin, right groin, both groins, spleen, stomach, intes-

tines, nose, nasopharynx, spine, right humerus, sternum, skin, heart, pericardium, pleura, lung and brain.

Additional symptoms are diarrhea or constipation, distention and diffuse abdominal tenderness. In some patients
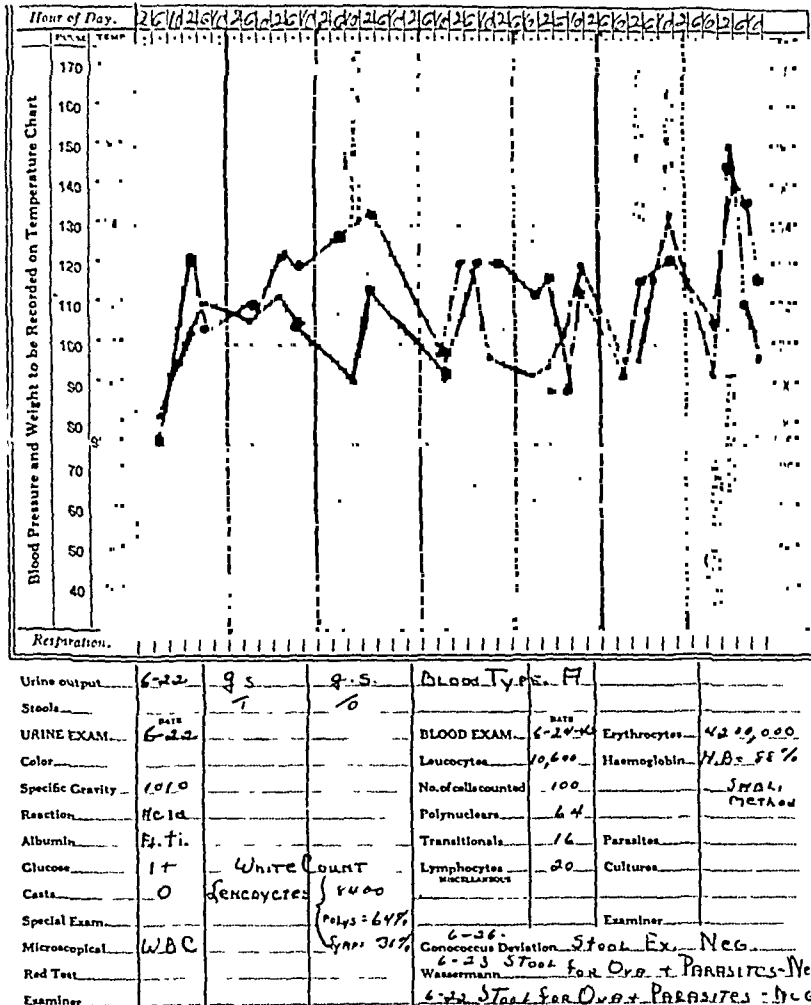
FIG. 2. Temperature chart for June 21st to June 27th.

Abdominal Hodgkin's disease presents great diagnostic and therapeutic difficulties, especially in cases in which there is no enlargement of the superficial lymph glands. In the early stages of the disease the symptoms are referable to the organ involved and in the absence of systemic symptoms, viz., fever, loss of weight, pruritus, etc., considerable time is lost before the diagnosis is made. Abdominal symptoms are common and in some patients they are the initial ones. Abdominal pain, simulating a gallbladder syndrome or peptic ulcer, is not uncommon.

there may be generalized brownish pigmentation, or the discoloration may be irregularly distributed, giving a mottled appearance. Occasionally, there may be an actual infiltration of the skin, producing small nodules. An extreme degree of pruritus may be an early and annoying symptom which sometimes constitutes the patient's chief complaint. The disease begins as a localized process and the prognosis depends on irradiation before generalization takes place.

In Hodgkin's disease of the small intestine, it should be recalled that scattered

over the mucous membrane of the entire
length of the intestine there are numerous
minute, soft, rounded bodies composed of

Where lymph-nodes are accessible, if
excised and followed immediately by roent-
gen irradiation, this combined treatment
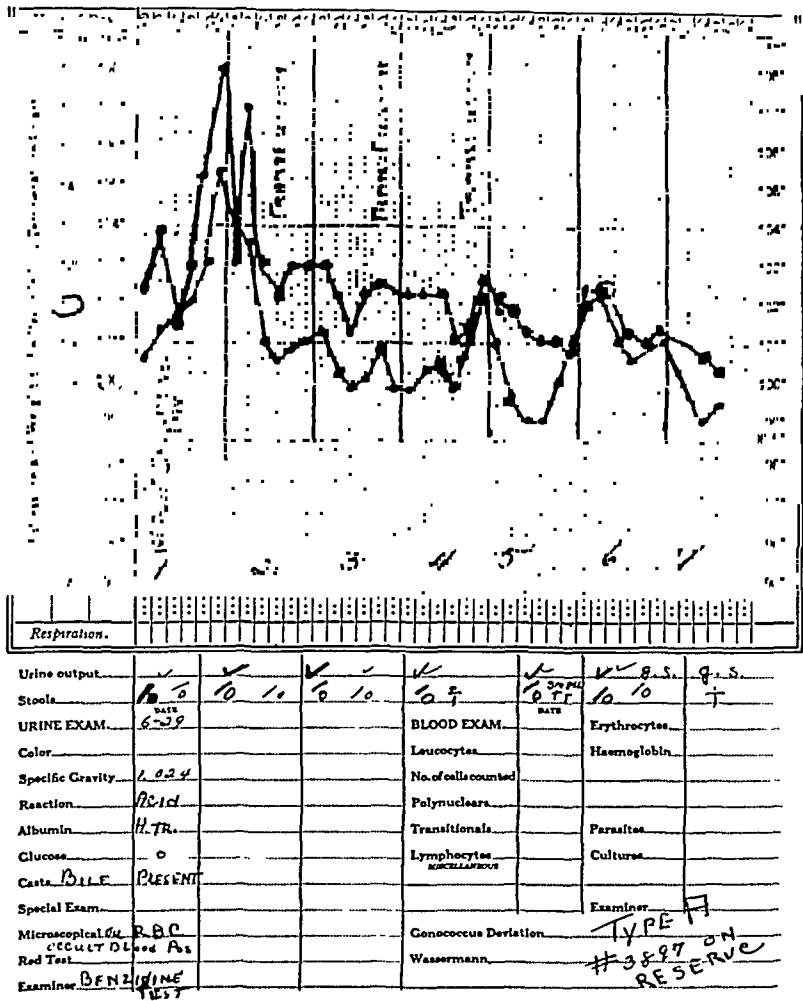
FIG. 3. Temperature chart for June 28th to July 4th.

retiform tissue, constituting the solitary
glands. The aggregation of these solitary
glands form Peyer's patches. These patches
are more numerous in the lower ileum, and
it is in these lymphoid structures that the
disease affecting the small intestine most
likely originates.

X-ray examinations offer invaluable aid
in diagnosis. It helps to determine the seat
and extent of certain deep lesions; it
aids in the verification of the regression
after treatment and the extension after
recurrence.

seems to give better results. Most observers
prefer methodical radiotherapy. In their
opinion it brings about pronounced remis-
sions and prolongs life.

CASE REPORT

C. S., a white female twenty-nine years old,
para I, with a child sixteenth months old and
well, had never been acutely ill in her life. Six
months before she began to have attacks of
diarrhea, the daily movements averaging from
six to eight per day. Shortly after she began to
complain of anorexia, weakness and loss of
weight. The attending physician treated her

as a case of incipient tuberculosis. Three months later besides the enumerated symptoms, fever of low grade made its first appearance. Other physicians were called on the case and in spite of negative x-ray finding of the chest and that there was no cough present, the treatment was continued along the lines of pulmonary tuberculosis. In the fourth month of her illness, the patient was interned in a hospital, where she remained for a month and was discharged with the diagnosis of ulcerative colitis. The patient came to the author's attention who had her admitted to Morrisania Hospital with the presumptive diagnosis of degenerating diverticulitis.

The patient was chronically ill, markedly emaciated and complained of anorexia, diarrhea, asthenia, loss of weight, fever, with afternoon excursions reaching from 103–104°F.

There were no sweats but there were left upper and middle abdominal quadrant pains, associated with vomiting twenty to thirty minutes after meals. There was no history of hematemesis, melena or mucus in the stools no hematuria, pruritus, no cyanosis, dyspnea, cough or jaundice. Scattered over the forehead and cheeks there were areas of dark pigmentation. Superficial lymph-nodes were normal in appearance and distribution. The lungs produced crackling râles over the fourth and fifth ribs anteriorly. The abdomen was not as emaciated as the rest of the body; tenderness was present in all the quadrants, but in the left upper and middle quadrants, tenderness was greater and an ill defined mass could be palpated there. Vaginal and protoscopic examinations showed no abnormality. Blood examination revealed 12,600 white blood cells, 64 polymorphonu-

FIG. 4. Temperature chart for July 5th to July 11th.

clears, 16 transitional, 20 lymphocytes, hemoglobin 88 per cent. This blood examination was repeated several times, looking for eosinophilia

to 5 Gm. of sulfanilamide was thrown in the region of the anastomosis and the abdomen was closed in layers without drainage.

A   B

FIG 5. A, showing inner surface with eroded area; B, posterior view of jejunum with eroded area.

and for any informative material. The patient at this time had received many transfusions and that in itself would have somewhat altered the true blood picture. Blood sedimentation was 16; blood cultures were negative, also for Brucella. Stools were negative for tuberculosis organisms, ova or parasites. Urine and Widal tests were negative.

On June 26, six days after admission, a laparotomy was performed with a left rectus incision. Operative findings disclosed a loop of thickened inflamed jejunum, with many verruca-like excrescences, 1 to 2 mm. in diameter on the serosa. The loop, horse-shoe shaped, had large mesenteric glands in its concavity and the convex portion of the loop was plastered and covered by the omentum and transverse colon. There was no free fluid or fibrin seen in the peritoneal cavity. There was no distention of the small bowel or colon proximal to the lesion. The affected loop with blunt dissection was gently separated from the omentum and the colon. The colon did not show much gross pathological disturbance but on the convex surface of the loop there was an eroded area about 2 cm. in diameter; evidently a slow perforation of the jejunum had taken place and the omentum with the colon had been supporting the gnawed jejunal wall. About 20 cm. of what appeared to be diseased jejunum was resected and a side-to-side anastomosis was performed. The mesentery was studded with glands averaging from pea to walnut in size and considerable bleeding was encountered in ligating the mesenteric vessels. The mesentery, evidently made up of merging tumor tissue, was very friable. Three

The patient was a poor surgical risk. The morning of the operation she had a temperature of 104°F. and the postoperative reaction brought the temperature to 106°F.

Macroscopic examination revealed the small intestine to be about 20 inches long, the wall markedly thickened, varying from $\frac{1}{2}$ to 1 inch, the lumen considerably dilated, mucosa hypertrophic; one area was necrotic and appeared constricted.

Biopsies taken from various portions of the resected loop showed similarity of structure, which was characterized by complete obliteration of normal structure from mucosa to serosa with diffuse replacement by lymphomatous tissue. The cells were variable in size and appearance, showing hyperplasia of reticulum cells and lymphocytes. Numerous giant cells of the Reed-Sternberg type were scattered throughout. There were a number of areas of necrobiosis and slight fibrosis. Superimposed polynuclear exudate was present in the more degenerated areas. The entire structure was one of granuloma with abundant histocytes and Reed-Sternberg cells.

Postoperatively the temperature curve remained the same. The wound healed per primum. She received daily doses of arsenic parenterally. She was given transfusions every fourth or fifth day. On July 23, the abdomen was divided into quadrants and she was daily subjected to roentgen therapy for thirteen days; the patient at this time was too weak and the therapy was discontinued. On August 7, the patient had two bloody bowel movements. On August 8 she had another bloody bowel movement. On August 14, her temperature

rose to 105°F. and the patient expired on the forty-ninth postoperative day.

COMMENTS

1. The illness of this patient was a diagnostic puzzle from the beginning. Perhaps by a process of elimination, the diagnosis could have been made.

2. In abdominal Hodgkin's disease, in the absence of enlargement of the superficial nodes, a small spleen and no leading symptoms, one doubts whether the diagnosis could have been made.

3. During the short interval prior to the operation considerable work was done; further study was abandoned because the patient was rapidly going downhill, and in the presence of a mass in the left upper and middle quadrants, an immediate exploratory was decided upon.

4. The diarrhea and the pigmentation on the face were not advantageously evaluated.

5. The pyrexia (never of the Pel-Ebstein type), the malignant course (with the patient losing seventy pounds in seven months), the extensive pathological involvement, leads one to classify this case as a malignant Hodgkin's sarcoma.

6. Forty days after the operation, the patient had bloody stools on several occasions, leading us to believe that further intestinal involvement had taken place in proximity to the anastomosis.

7. Following the high voltage roentgen therapy there was neither subjective nor objective improvement. Perhaps the treatment was instituted too late.

8. This case maintained an elevated pulse rate, completely out of proportion to the temperature, which according to L. Goldman in a series of 212 cases, is a striking feature.

9. In this advanced type of Hodgkin's disease with numerous glands in the mesentery, surprisingly, the spleen remained small There was neither eosinophila present nor pruritus.

10. The sedimentation rate in this case repeated several times maintained an average of 14 to 16 instead of a higher sedimentation curve which one would expect in this advanced type of Hodg-
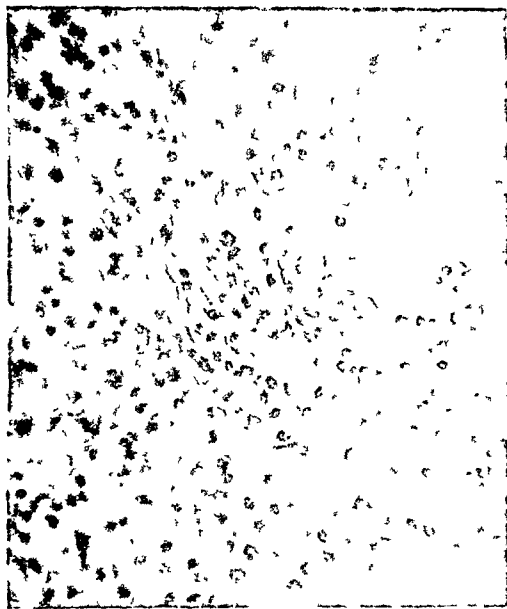
FIG. 6. Photomicrograph of the tissue showing multinuclear giant cells.

kin's disease, perhaps influenced by the transfusions.

11. The secondary cause of death here must have been terminal exhaustion, anemia and the pyrexia which had been present for four months.

CONCLUSION

This case had several diagnostic symptoms which although not pathognomonic were very suggestive of Hodgkin's disease: the prolonged diarrhea without a specific cause, the marked asthenia from the beginning of her illness, loss of weight, anemia, pyrexia, abdominal pain, elevation of her pulse out of proportion to the temperature and pigmentation (though localized on the face). When all these facts are correlated with the pathological findings and evaluated, the picture of Hodgkin's disease would stand out more prominently.

Through the courtesy of the Second Surgical Division of Morrisania City Hospital, headed by Dr. Peter T. Daly, the above case report is offered.

REFERENCES

HODGKIN, T. *Med. Chir. Tr.*, London, 17: 68, 1832.

GARVIN, F. C. Hodgkin's disease of the heart and pericardium. *J. A. M. A.*, 117: 1876, 1877.

STERNBERG, C. *Ztschr. f. Heilk.*, 19: 21, 1898.

REED, D. *Bull. Johns Hopkin's Hosp.*, 10: 133, 1902.

PRIESEL, A. and WINKELBAUER, A. *Virchows Arch. f. path. Anat.*, 262: 749, 1926.

MINET, G. R. and ISAACS, R. Lymphoblastoma. Aspects concerning abdominal lesions. *Am. J. Med. Sc.*, 172: 157, 1926.

MINET, G. R. and ISAACS, R. Lymphoblastoma (malignant lymphoma). *J. A. M. A.*, 86: 1185, 1926.

GORDON, M. H. Rose research on lymphodenoma. Bristol, 1932. John Wright & Sons Ltd.

SMITH, C. S. *J. Pediat.*, p. 412, 1934.

PEIRCE, C. B. JACOX, H. W. and HILDRETH, R. C. *Am. J. Roentgenol.*, 36: 145, 1936.

EDWARD, D. S. *Lancet*, 1: 936, 1938.

McNAUGHT, J. B. Gordon test for Hodgkin's disease; reaction to eosinophils. *J. A. M. A.*, 111: 12181, 1938.

TURNER, J. C., JACKSON, H. JR. and PARKER, F. *Am. J. Med. Sc.*, 19: 527, 1938.

FINZI, N. S. Roentgen treatment of lymphodenoma. *Am. J. Roentgenol.*, 39: 261–262, 1938.

EPSTEIN, E. Sex as factor in prognosis of Hodgkin's disease. *Am. J. Cancer*, 35: 230–233, 1939.

GOLDMAN, B. L. Hodgkin's disease. *J. A. M. A.*, 114: 1611–1616, 1940.

WISE, B. M. and POSTON, M. A. *J. A. M. A.*, 115: 1976–1984, 1940.

STEINER, P. E. Reliability and significance of Gordon Test on Hodgkin disease. *Arch. Path.*, 31: 1, 1941.

CHARR, R. and WASCOLONIS, A. Pulmonary lesions in Hodgkin's disease. *J. A. M. A.*, 116: 2013 and 2014, 1941.

WINKELMAN, N. W. and MOORE, M. T. Hodgkin's disease of the nervous system. *Arch. Neurol. & Psychiat.*, 45: 304–317, 1941.

SLAUGHTER, D. P. and CRAVER, L. E. *Am. J. Roentgenol.*, 7: 596–606, 1942.

IT is a well-known observation that, after parathyroidectomy, both in animals and in man, the hair may fall out in large amounts over a period of some months. The reason is not clear but it has been suggested that it is connected with disturbances of mineral metabolism.

From "Symptoms in Diagnosis" by Jonathan Campbell Meakins (Little, Brown and Company).

CARCINOMA OF BARTHOLIN'S GLAND

THELMA G. BOUGHTON, M.D.

Assistant Pathologist, Lancaster General Hospital

LANCASTER, PENNSYLVANIA

CARCINOMA of Bartholin's gland is very rare and probably has often been a missed diagnosis. The earliest reference to a malignancy of this gland was made by Klob in 1864. In his book on the pathological anatomy of the female sex organs he mentioned, in connection with chronic inflammation of Bartholin's gland, a tumor formation which he described as resembling a cystosarcoma of the breast. He thought that this malignancy resulted from the chronic inflammation. In this same book, cysts of Bartholin's gland and carcinoma of the vulva are discussed separately with reference to the earlier literature but a definite, indisputable carcinoma of Bartholin's gland is not described. Since that time occasional reports of one to three cases have appeared. Several authors, especially the German and English, have also reviewed the literature, collected the known cases, summarized the data and incidentally have provided extensive bibliographies. The most complete and most valuable of these studies have been made by Honan (1897), Sitzenfrey (1906), Spencer (1914), Wittkopf (1915), Falls (1923), Harer (1933), Rabson and Meeker (1938), Gobel (1938) and Simendinger (1939).

The exact incidence of this tumor is difficult to determine. Many of the reported cases, especially the earlier ones, are incomplete and the details essential for verification of their diagnosis are lacking. It is also obvious that many of these cases are reported as carcinomas of the vulva rather than of Bartholin's gland because they are seen too late and when the growth is too extensive to permit determination of their exact origins. Another factor which contributes to the uncertainty as to the actual incidence of this tumor is the possible total lack of diagnosis in early cases removed without subsequent laboratory study. This possibility is suggested by the fact that, in many reported cases, the true nature of the lesion was discovered only at operation or when the material was examined by a pathologist. Unless all cysts and abscesses of Bartholin's glands are made the subject of careful pathologic study, the incidence of its carcinoma in its early stages will be uncertain. Rabson and Meeker, in 1938, reported a collection of fifty-six cases from the world literature and added two of their own. In the same year, Göbel collected fifty cases and added three. In May, of 1939, Simendinger added eighteen cases to a list of twenty made in 1923 by Falls. These two authors attempted to list only the bona fide cases and to give comparative data as regards age of patient, type of tumor found, metastases, treatment and results. Simendinger had four cases, including his own, which Rabson and Meeker had not reported. In November, of 1940, Cosbie reported one additional case which was included in his study of fifty-nine cases of vulvar carcinoma. In this same month, Taussig published a report of his experience with 155 cases of vulvar carcinoma, nine of which originated in Bartholin's gland. It is evident that although there are about seventy-five cases reported in the literature, a great number of these are published with inadequate data and may not be accepted as proved cases of Bartholin gland carcinoma.

This infrequent occurrence of Bartholin gland carcinoma is also reflected in textbooks and treatises on gynecology where it is usually mentioned in connection with the discussion of carcinoma of the vulva. It is given the same course and the same

bad prognosis which would be expected when it is so often seen only in its advanced stages after the skin of the vulva has been

FIG. 1. Low power of a section of a Bartholin's gland showing chronic inflammation with squamous cell replacement of its columnar epithelium.

FIG. 2. High power of the same section showing detail of stratified squamous epithelium replacing the columnar epithelium of the gland ducts.

involved. But this tumor is worthy of separate consideration, at least in regard to its early diagnosis and prognosis. Although Bartholin's gland is a part of the vulva, it is a distinct encapsulated structure having a well developed stroma. It gives rise to a carcinoma which, in its early stages especially before the capsule is penetrated, would be expected to give a greater hope for cure than that of the vulva where a malignancy so often arises on an already altered epithelium (as in leukoplakia) and may be multicentric. The data published do not prove this to be true; but in view of the fact that so many of the cases are seen after the patient has realized a lump in the labium for a period of six months or several years, or, as in a few cases, the lesion has been treated unsuccessfully as a Bartholin's abscess, including incision and drainage of necrotic tissue, there is reason to ask whether these might not permit a hopeful prognosis if completely removed and diagnosed early.

Honan, in 1897, suggested criteria for the diagnosis of Bartholin's gland carcinoma and reference to these is repeatedly made in the literature. He suggested the diagnosis whenever there is a tumor mass in the typical site of Bartholin's gland, a tumor deep in the labium especially if the overlying skin is intact, a tumor having a microscopic picture bearing some resemblance to the normal gland epithelium and the presence within the tumor of normal gland tissue or its connection with the gland duct. Clinically, it can be suspected with the presence of a lump in the region of Bartholin's gland, i.e., the posterior third of the labium majus. The lump is usually reported as hard, sometimes nodular, sometimes freely movable or sometimes bound to adjacent structures. If there is necrosis of tissue the tumor may appear to be cystic or soft and this is the condition which leads to its treatment for abscess with a diagnosis of carcinoma after the lesion has been incised and fails to heal. In the early stages there are usually no subjective

symptoms and neither the patient nor the physician is impressed with the seriousness of the situation. Taussig advised close

makes possible the origin of either squamous cell or adenocarcinoma within the deeper portions of the gland.
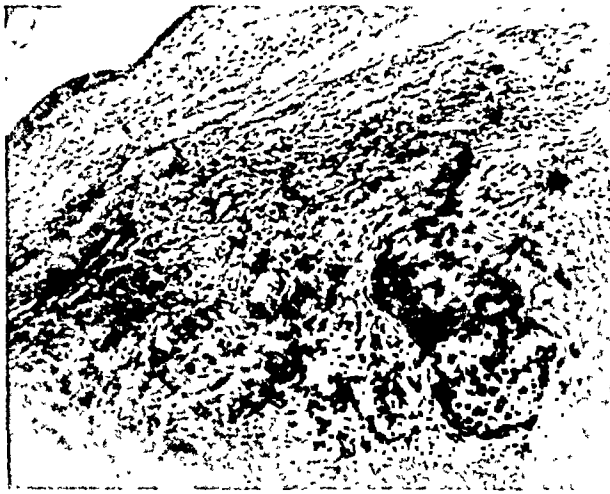
FIG. 3. Squamous cell carcinoma of Bartholin's gland seen invading the wall of the duct. The duct wall is shown in the upper left corner of the photograph.

observation or complete excision of enlarged Bartholin's glands in all women over forty years of age.

The normal epithelium of this gland has been repeatedly described. It is generally agreed that columnar epithelium lines the alveoli and that in the ducts there is epithelium of a transitional type composed of cylindrical cells in many layers. This is quite characteristic of Bartholin's gland and is not simulated by any other gland structures of the labium. Stratified squamous epithelium is found normally only at the orifice and in the terminal portion of the main excretory duct, but in chronic inflammatory conditions of the gland, several authors have described the occurrence of this type of epithelium in the deeper parts. Sitzenfrey was the first to illustrate this condition and he was followed by Schweitzer who called it metaplasia. In the surgical specimens of our laboratory this phenomenon has been repeatedly observed. It presents itself as a replacement of the cylindrical or transitional epithelium by the stratified squamous type in the manner of that which takes place in chronic inflammation of the cervix uteri. (Figs. 1 and 2.) This condition

Extensive search of the literature reveals no special study of the lymphatics which drain Bartholin's gland; they are generally considered to be the same as those from the vulva. After a detailed study of the lymph-nodes involved in cases of vulvar carcinoma, Taussig has divided them into five groups: the inguinofemoral group, those of the external iliacs, the obturator glands, the hypogastric glands and the ureteral glands. In sixty-five cases of vulvar carcinoma there were thirty with lymph-node metastases. The inguinofemoral chain was involved forty times, twenty-one times on the right and nineteen times on the left. In only five cases were the deeper glands involved, the right external iliacs twice, the right obturators once, the left external iliacs once and the left obturators once. The conclusion is drawn that most metastases from vulvar carcinomas occur in the inguinofemoral chains on either or both sides and that rarely is there a metastasis into the deeper nodes. The application of this information to the lymphatic spread of Bartholin's gland carcinomas is open to question, especially those arising in the deeper portions of that structure.

The therapy advised for Bartholin's gland carcinoma is varied as would be expected when few surgeons have had experience with more than one to three cases. In most cases reported there is a tendency to do a radical excision with removal of the inguinofemoral glands of both sides because of the lymphatic anastomosis in the mons veneris. Few attempt to remove the deeper nodes because if these are involved the case is hopeless. Hunt and Powell discussed this therapy in their paper of 1926 and questioned the advisability of ever removing the lymph-nodes. Probably the only successful treatment is early, complete excision of the tumor.

It is because of the rarity of this tumor, the fact that if kept in mind its presence is readily suspected and because of the belief that if diagnosed and removed early there may be hope for its cure, that this report is written.

CASE REPORT

I. H., a white woman, age fifty-four, was admitted to Lancaster General Hospital on June 6, 1935, with the following complaints: swelling in the vagina and rectum, painful bowel movements and itching of the anus. Hemorrhoids had been noticed for two years. For several years the patient had realized that her vagina was getting smaller and for six to seven months she had noticed a swelling inside and beside the vagina and the rectum. There was slight pain on defecation. No bleeding had occurred but there was a slight vaginal discharge. Itching of the anus, which brought the patient to the physician, had persisted for three weeks. She had also suffered a weight loss of fifteen pounds in four to five weeks.

The past history was irrelevant except for chronic pelvic inflammatory disease of many years' duration and for which she was operated in 1925. At that time the Fallopian tubes which were bound down in the cul-de-sac were removed. A thorough pelvic examination then revealed no evidence of tumor.

On June 8, 1935, the patient was operated upon. A tumor which extended from the region of the vagina into the rectovaginal space and back into the ischiorectal space was removed.

The overlying epithelium in both vagina and rectum was removed along with the tumor because of gross changes which made its involvement seem probable. An attempt to repair the pelvic floor and the anal sphincter was made. No enlargement of the inguinal nodes was found at that time.

The tumor was examined and its pathology reported by Dr. Louisa E. Keasbey of this hospital. The specimen weighed 67 Gm. and measured 6 by 4.5 by 4.5 cm. It appeared to be irregularly dissected from the surrounding tissue and did not seem to be completely removed. The mass was surmounted in one area by rectal mucosa which was not thickened and appeared grossly not to be the source of the tumor. Beneath the mucous membrane, the muscular coat of the rectum was identified but was incomplete. An area of vaginal mucosa was present and also appeared not to be involved by the tumor process. The tumor had a fibrous texture.

Microscopic examination showed broad sheets and masses of squamous cell carcinoma infiltrating a rather abundant fibromuscular stroma. The masses of tumor cells were exceedingly large and anastomosed with one another. They showed hornification. The differentiation between the periphery and the centers of these masses was ill defined and irregular. In the centers large areas of hyaline degeneration were found. The carcinoma cells varied markedly in size and shape. Those in the centers were large and round; the peripheral ones were smaller and fusiform. Many giant cells and bizarre nuclei were present. Mitotic figures averaged about 6 per 10 high power fields. The stroma showed little evidence of inflammation. The vaginal mucous membrane present was not involved and microscopically a broad band of fibrous tissue separated the tumor from the rectum. In certain areas the carcinoma surrounded an empty branching lumen as would a neoplasm spreading along the walls of a duct. The alveoli resembling those of Bartholin's glands were compressed and atrophic. They did not participate in the carcinomatous process. At the periphery of the carcinoma in certain sections, compressed tubules of the type seen in Bartholin's glands were found and the impression was gained that this tumor arose in the duct of Bartholin's gland.

The postoperative course was uneventful except for the fact that fecal incontinence

persisted and on July 24, a colostomy was done. The patient then made a good recovery and on August 8, 1935, she left the hospital

notice blood in her urine. She appeared to be in good condition and the colostomy bag was functioning satisfactorily. Cystoscopic exam-
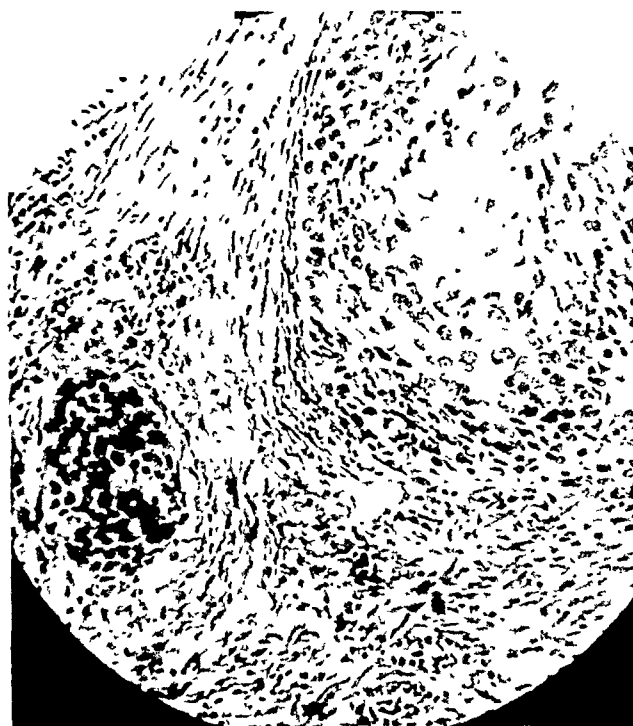
Fig. 4. High power field showing detail of the squamous cell carcinoma.

wearing a colostomy bag which was functioning well.

Postoperative deep x-ray therapy was begun on June 28 and continued until December 20. Four portals encircling the pelvis were used with the following factors: 20 K.V. and filters of 0.5 mm. copper with 2 mm. aluminum at a distance of 50 cm. The portals each measured 15 by 15 cm.

On June 26, 1937, the patient returned with pain in the right hip. There was no history of trauma. The pain had started four weeks previously and had become progressively worse until she was unable to walk. X-ray showed a fracture of the neck of the femur without displacement. Considering the possibility of a pathological fracture following metastasis an x-ray examination of the lumbar spine, the entire pelvis and both femora was made. No evidence of tumor was found. With proper treatment the patient recovered and returned to her former state of good health.

On November 11, 1939, the patient returned again. She had been very well until about three weeks previously when she began to

ination showed a severe inflammation of the bladder which was treated.

On August 16, 1940, she returned complaining of bleeding from the rectum. There was slight bleeding from the distal loop of bowel after irrigation. On physical examination she was found to be fairly well nourished and suffered no discomfort. Both abdominal and anal stomas were too strictured to admit the examining finger. Vaginal examination showed a firm mass about the size of a peach in the rectovaginal septum and the perineum with some induration forward around the vaginal introitus on the right side. The induration was possibly scar from the original resection but the main mass seemed probably to be a local recurrence of the tumor. No evidence of distant metastasis was found. On August 29, 1940, an abdominoperineal resection of the rectum and sigmoid with the entire posterior vaginal wall was done. The wide wound was repaired as much as possible.

Examination of the tissue removed at operation showed a squamous cell carcinoma resembling that which was first removed except

for the presence of much more pleomorphism and a scanty amount of stroma. It extended to the mucous membrane of the rectum and the vagina.

Postoperatively the patient reacted well and progressed satisfactorily until September 24 when she began to have a high fever and blood in her urine. This condition persisted in spite of treatment. She failed gradually and expired on November 9, 1940.

Autopsy was performed on the day of death. There was evidence of marked weight loss. The sigmoidostomy wound was intact but was associated with a small sinus tract that extended into the subcutaneous tissue of the abdominal wall. The perineal wound appeared clean and its granulation tissue bled easily. There was a patchy pneumonia especially marked in the right lung. The peritoneal cavity was clean but a pyelonephritis was found. This was more marked in the right kidney where the urine seen in the pelvis was frankly purulent. A thorough search grossly and microscopically was made of the pelvic and inguinal lymph-nodes and no evidence of tumor metastasis was found. Sections were made of the wound edge looking for areas of tumor tissue and none were found.

CONCLUSIONS

1. Carcinoma of Bartholin's gland is rare if its incidence is judged by the number of cases reported. Its exact incidence is uncertain because of the fact that many cases are reported as carcinoma of the vulva without discrimination as to definite origin, and that many cases are reported as carcinoma of Bartholin's gland without adequate data to prove this fact.

2. The prognosis, according to the literature in which it is often classed as a carcinoma of the vulva, is poor. However, a study of its distinct anatomic structure and a consideration of the fact that many cases are seen when the lesion is far advanced suggests that the prognosis might be more hopeful. A review of the case reported in this paper supports the more favorable prognosis in that this tumor, although seen after a duration of

several years, showed no metastases in over five years following its excision.

3. No study of the lymphatic drainage of Bartholin's gland, distinct from that of the vulva, is found. Carcinoma of the vulva is reported to have involved the regional lymph-nodes in less than half of the cases and then usually the inguino-femoral chains. The application of this information to lymph drainage from carcinoma of Bartholin's gland is probably open to question.

4. If kept in mind, the presence of this tumor may easily be suspected in its early stages with hope that prompt complete excision may affect a permanent cure.

I wish to express my appreciation and thanks to Dr. S. G. Pontius and Dr. Ross Proctor for the use of their clinical data of this case.

REFERENCES

1. BECKMANN, GEORG. Adenokarzinom der Bartholinischen Drüse in jugendlichen Alter. *Dermat. Wchnschr.*, 100: 101, 1935.
2. BERKELEY, C. and BONNEY, V. Leukoplakic vulvitis and its relation to kraurosis vulvae. *Proc. Roy. Soc. Med. (Sect. Obst. & Gynec.)*, 3: 29, 1909.
3. BLAIR-BELL, W. and DATNOW, M. M. Primary malignant disease of the vulva, with special reference to therapy by operation. *J. Obst. & Gynaec., Brit. Emp.*, 43: 755, 1936.
4. CHATON, M. Observation d'un cas d'epithelioma primitif de la glande Bartholin. *Bull. Assoc. franç p. l'etude du cancer*, 26: 585, 1937.
5. COSBIE, W. G. Carcinoma of the vulva. *Canad. M. A. J.*, 43: 439, 1940.
6. DATNOW, MORRIS M. Discussion on carcinoma of the vulva. *Proc. Roy. Soc. Med.*, 32: 298, 1937.
7. DAVIS, JAMES E. The pathology of the major vestibular ducts and glands. *Am. J. Obst.*, 75: 58, 1917.
8. DE ROUVILLE, G. Epitheliome atypique paravaginal développé fort probablement aux dépens d'une glande de Bartholin. *Ann. de gynec.*, 42: 569, 1916–1917.
9. DITTRICK, H. Epithelioma of the vulva. *Am. J. Med. Sc.*, 130: 277, 1905.
10. EWING, JAMES. Neoplastic Diseases. 4th ed., p. 621. Philadelphia, 1940. W. B. Saunders Co.
11. FABRICUS, J. Berichte aus Gynakologischen Gesellschaften und Krankhausen. *Zentralbl. f. Gynäk.*, 33: 995, 1909.
12. FALLS, F. Carcinoma of Bartholin's gland. *Am. J. Obst. & Gynec.*, 6: 673, 1923.
13. GÖBEL, A. Clinical and therapeutic study of carcinoma of Bartholin's gland. *Zentralbl. f. Gynäk.*, 62: 1970, 1938.

14. GRAHAM, JAMES M. Cancer originating from Bartholin's gland. *Edinburg M. J.*, 23: 149, 1908.

15. HARER, W. BENSON. Primary carcinoma of Bartholin's gland. *Am. J. Obst. & Gynec.*, 25: 714, 1933.

16. HONAN, J. H. Ueber die Carcinome der Glandulae Bartholini. Inaugural Dissertation, Berlin, May 28, 1897.

17. HUNT, V. and POWELL, L. D. Malignant disease of Bartholin's gland. *Surg. Clin. North America*, 6: 1325, 1926.

18. KELLY, H. A. Operative Gynecology. 2nd ed., vol. 1, p. 258. D. Appleton & Company, New York, 1906.

19. KLOB, JULIUS M. Pathologische Anatomie der weiblichen Sexualorgane. P. 471. Wien, 1864. Wilhelm Braumüller.

20. LESTRADE, M. Contribution a l'etude des tumeurs solides de la glande de Bartholin. *Arch. med. de Toulouse*, 7: 265, 1901.

21. LYLE, H. H. M. Primary carcinoma of Bartholin's gland. *Ann. Surg.*, 100: 993, 1934.

22. LYNCH, F. W. and MAXWELL, A. E. Pelvic Neoplasms, P. 419. New York, 1931. D. Appleton & Company.

23. MAYO, C. W. and BARBER, K. W. Carcinoma of Bartholin's gland. *Surg. Clin. North America*, 14: 709, 1934.

24. MILLAR, WM. M. and LANDEN, MINNIE. The tumor clinic of the Cincinnati General Hospital—a record of its first decade. *Ohio State M. J.*, 35: 1069, 1939.

25. NEUWIRTH, KARL. Ein weiterer Beitrag zum Studium des Karzinoms der Bartholinischen Drüse. *Monatscbr. f. Geburtsb. u. Gynäk.*, 70: 93, 1925.

26. POLAILLON. Affections chirurgicales du trone, mammelles, organs génitaux de la femme, P. 147. Paris, 1901. Octav Doin.

27. RABINOVITCH, J. Carcinoma of Bartholin's gland. *Am. J. Obst. & Gynec.*, 23: 268, 1932.

28. RABSON, S. M. and MEEKER, L. H. Carcinoma of the major vestibular (Bartholin) gland. *Surg., Gynec. & Obst.*, 76: 505, 1938.

29. ROSAHN, P. D. Summary of the experiences with malignancies at the New Britain General Hospital, 1929–1938. *J. Connecticut M. Soc.*, 3: 405, 1939.

30. SCHREINER, B. F. and WEHR, W. H. Cancer of the vulva with a report of 118 cases. *Surg., Gynec. & Obst.*, 58: 1021, 1934.

31. SCHULZ, KURT. Zur Kasuistik und Statistik des Vulva Karzinom. *Centralbl. f. Gynäk.*, 56: 2364, 1932.

32. SCHWEITZER, F. Carcinoma glandulae Bartholini. *Arch. f. Gynäk.*, 44: 322, 1893.

33. SIMENDINGER, E. A. Carcinoma of Bartholin's gland. *Surg., Gynec. & Obst.*, 68: 952, 1939.

34. SITZENFREY, D. A. Hornkrebs des Gangsystems der Bartholinischen Drüse. *Ztscbr. f. Geburtsb. u. Gynäk.*, 58: 24, 1906.

35. SPENCER, H. R. Case of primary carcinoma of Bartholin's gland. *Proc. Roy. Soc. Med.*, 7: 102, 1913–1914.

36. STRAUSS, H. Carcinoma of Bartholin's gland. *J. A. M. A.*, 101: 2116, 1933.

37. TAUSSIG, F. J. Cancer of the vulva—an analysis of 155 cases. *Am. J. Obst. & Gynec.*, 40: 764, 1940.

38. TAUSSIG, F. J. A study of the lymph glands in cancer of the cervix and cancer of the vulva. *Am. J. Obst. & Gynec.*, 36: 819, 1939.

39. WITTKOPF, H. Ueber das Karzinom der Bartholinischen Drüse. *Zentralbl. f. Gynäk.*, 39: 369, 1915.

SUCCESSFUL PRIMARY REPAIR OF LACERATED PAROTID DUCT

EDWARD A. CHRISTOFFERSON, M.D.,
Attending Surgeon, Cook County Hospital

METRY PETER AJALAT, M.D.
First Lieutenant, U. S. Army

AND

RALPH GRADMAN, M.D.
First Lieutenant, U. S. Army
CHICAGO, ILLINOIS

ALTHOUGH laceration of the parotid duct is not a very common occurrence in peace time, it may become relatively frequent during the course of the present war. This expected increase in frequency brings the necessity for the proper care of such wounds into prominence. This is particularly true when we remember the high incidence of development of external fistulas in mismanaged cases and the prolonged morbidity which results from such complications.

In reviewing the literature, we find that only five successful repairs have been reported. It is perhaps significant to note that the first case was reported by Dr. Tees[1] in 1926. He used catgut as a dowel in the duct and left the end projecting in the mouth. He then approximated the ends of the duct with two fine interrupted catgut sutures passed through the sheath around the duct. In 1927, Dickinson[2] used the same technic but used silkworm gut for a dowel. He fixed it in the buccal mucosa by a suture. However, the suture fell out in two days. Black and Flagge,[3] in 1928, used a small ureteral catheter as a dowel and left one inch projecting into the mouth, and fixed it by interrupted linen sutures around the opening of the duct. The catheter was removed on the ninth postoperative day. Brohm and Bird,[4] in 1935, used a filiform ureteral bougie as a dowel. They passed the distal end of the bougie through the cheek and sutured it to the skin at the point of exit. In 1933, Butler and Guinan[5] reported a successful repair forty-one days after the injury. They used a ureteral catheter as a

dowel and anchored the distal end to the upper canine tooth with a silk ligature. This arrangement allowed slight motion of the catheter in the duct when the lower jaw moved. Butler believes that this motion is advantageous. We, however, believe that such an arrangement is injurious because of the trauma to the delicate mucosa of the duct produced by the motion. This is confirmed by the fact that the authors were forced to remove the catheter on the third postoperative day because of the swelling.

It is perhaps interesting to note, that with slight variation all of these men used the same technic for the repair of the duct. However, there was diversion of opinion as to the type of dowel and the method of fixation used, most of which required special instruments. In our case, we used a very simple method which can be easily adapted to army conditions. This was done in our minor surgery room with morphine sulfate and scopalamine the only anesthetic used.

CASE REPORT

A. C., a twenty-seven year old colored female, was in an auto accident and was admitted to the hospital on January 16, 1942, five and one-half hours after the accident. She received several lacerations and abrasions of the face. The most prominent laceration was one midway between the right angle of the mouth and the tragus of the right ear. It was about five inches long and extended from the inferior angle of the mandible straight upward. The parotid duct, the branches of the facial nerve and the masseter muscle were severed. The wound was cleansed with green soap and

water, irrigated with saline and débrided very carefully. The severed nerve fibers were too small to warrant repair. The cut ends of the parotid duct were then easily identified.

Repair. One end of a horsehair suture was placed in the lumen of the proximal end of the severed duct for a distance of about one inch and the other end of the horsehair was passed through the lumen of the distal end of the duct into the mouth. This was used as a dowel. The ends of the duct were then approximated by interrupted black silk sutures passed through the sheath around the duct. The wound was then closed in layers using No. o interrupted catgut for the deeper tissues and interrupted black silk for the skin. The fixation of the dowel was accomplished by taping it with adhesive tape to the skin of the angle of the mouth.

The postoperative course was uneventful. On the second postoperative day, salivary secretion was seen flowing from the parotid duct opening into the mouth. The dowel was removed on the seventh postoperative day and the patient was sent home on the ninth postoperative day and was referred to the Follow-Up Clinic, where she was seen at weekly intervals at first then once every two weeks. On the eightieth postoperative day, the patient was seen in the clinic. The wound was well healed with no induration and no evidence of the formation of a fistula. There was a normal salivary secretion on the affected side.

CONCLUSION

A case of traumatic laceration of the parotid duct with successful primary repair is described. We wish to emphasize what we believe are some of the important features in the management of these cases: The first essential is to transform a contaminated wound into a clean wound by thoroughly and gently cleansing with soap and water using cotton pledgets followed with saline irrigations. The ends of the duct must be carefully identified and handled gently so as to avoid further damage to the delicate wall. Then a horsehair dowel about six inches long is placed through each end of the duct so that it enters the proximal end about one inch, and allows the distal end to protrude through the mouth. The two ends of the duct are then approximated by one layer of interrupted silk sutures, placing the stitches through the outer sheath only. We advise the use of silk or any other equivalent nonabsorbable suture material, because the wounds heal by the so-called "dry healing" with a minimum amount of tissue reaction and there is lessening of subsequent stricture of the lumen of the duct. The dowel is best left *in situ* for at least seven days, since its function is merely to maintain patency and approximation of the lumen and to allow the secretions to pass between it and the duct wall. After seven days, its removel can be safely accomplished. It is essential that oral hygiene be maintained throughout the healing process, if secondary infection and parotitis are to be avoided.

REFERENCES

1. Tees, F. J. Primary repair of parotid duct. *Canad. M. A. J.*, 16: 155, 1926.
2. Dickinson, A. M. Repairs of injury to Stenson's duct. *New York J. Med.*, 27: 548–549, 1927.
3. Black, H. S. and Flagge, P. W. Successful anastomosis of Stenson's duct. *South. M. & Surg.*, 90: 755, 1928.
4. Brohm, C. G. and Bird, C. E. Primary repair of severed parotid duct. *J. A. M. A.*, 104: 733–734, 1935.
5. Butler, E. and Guinan, E. R. Successful repair of parotid duct. *Surg. Clin. North America*, 13: 1291–1293, 1933.

SURGICAL CONSIDERATION OF THE INGUINAL TRIANGLES

BENJAMIN H. BRUNKOW, M.D.

Billings Deaconess Hospital

BILLINGS, MONTANA

OVER the years much has been written about the anatomy and surgery of the inguinal region, so much so that seemingly there is nothing more to be said. However, occasionally the miner, on going over his old washings finds a nugget that he missed the first time. It is the purpose of this paper to call attention to two anatomical facts of this inguinal area, which if made use of will improve the operative results for repair of defects or deformities of this region.

Most instructors of anatomy teach the anatomy of the inguinal region as seen from the front which presents the abdominal wall anteriorally and ending at Poupart's ligament inferiorally. If one views the abdominal wall from within outward and along the lines of force which the abdominal contents exert on this region when the body is in the upright position, one gets a different picture. In this view it will be seen that Poupart's ligament is only an angle in the abdominal curve, the wall actually ending along the superior ramus of the pubis. Viewed from the side Poupart's ligament is the fascial band forming an obtuse angle with the abdominal wall and the superior ramus of the pubis. In planning for the repair of a hernia, the approach, at least mentally, should be from the inside and above Poupart's ligament at the origin of the trouble and not from the outside where one sees only the results of the internal pressure. The proper way to repair a hole in the dike is from the inside and not the outside.

Again viewing the abdominal wall from the inside (Fig. 1), one can see three triangles, one complete, the other two incomplete. The larger triangle which lies above Poupart's ligament is bounded medially by the obliterated umbilical veins, inferiorly by Poupart's ligament and laterally by the inferior epigastric vessels which traverse the subperitoneal space toward the tip of the uracus to join the medial side of the triangle.

Below Poupart's ligament there is a long narrow triangle with the base along the spine and ramus of the pubis; the apex at the inferior iliac spine and the sides being made up by the superior ramus of the pubis and Poupart's ligament. The triangle is filled by the iliapsoas muscle and the femoral nerve, artery and vein from out inward, respectively, except for a space about one inch long at the base. This space is normally filled with fat. This is the outlet through which a femoral hernia passes.

Crossing over Poupart's ligament in about its middle portion and lateral to the epigastric vessels is a cord-like structure, the vas deferens in the male and round ligament in the female, the former arising from the base of the bladder and the latter from the uterus, forming an acute angle about the inferior epigastric vessels and desending along Poupart's ligament (in the inguinal canal). It will be seen that the vas deferens or round ligament form a third triangle when viewed from the inside.

If one palpates forcibly against the abdominal wall, three areas will be felt: (1) the internal ring at the agress of the vas deferens in the male or the round ligament in the female, this being the site of an indirect hernia; (2) the base of triangle A, the site of a direct hernia; (3) at the base of triangle B, the site of a femoral hernia. These weak spots are all actually quite close together and in the cadaver can be

palpated by the thumb, index and middle fingers in apposition.

In the cadaver if one examines along the

FIG. 1. Structures about Poupart's ligament: (1) External oblique muscle and fascia; (2) lacuna exit of iliopsoas muscle; (3) Poupart's ligament; (4) iliopsoas fascia and septum; (5) internal inguinal ring; (6) femoral nerve, artery and vein; (7) upper portion of Cooper's ligament; (8) empty space, site of femoral hernia; (9) lower end of Cooper's ligament; (10) superior ramus of pubis; (11) vas deferens, pulled medially. Normally it takes a fairly direct course from the base of the bladder to the internal inguinal ring.

superior ramus of the pubis, the one side of triangle B, a well formed band of fascial tissue, Cooper's ligament, will be easily found. The function of this ligament is not definitely known but it has comparatively great strength. By passing a stout ribbon around it, the cadaver may be suspended by this structure alone. Poupart's ligament can be torn with much less force. Cooper's ligament seemingly was just placed in this position to facilitate a strong anatomical hernia repair.

PRACTICAL APPLICATION

In the repair of hernia, regardless if it be indirect, direct inguinal or femoral, it should be the surgeons endeavor to eliminate all three of these weak spots, not just one, to guard against another hernia in this region. The basis of most hernia

repair is the suturing of the conjoined tendon, or reflected ligament, to the shelving edge of Poupart's ligament. This is
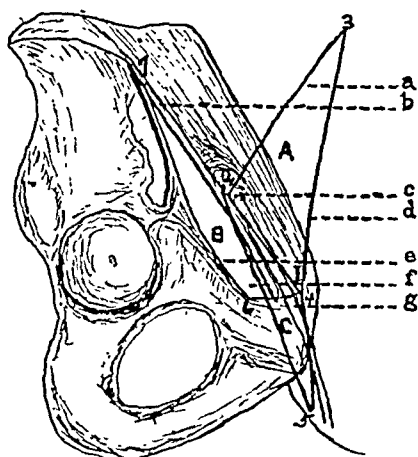
FIG. 2. Inguinal triangles semidiagrammatic: (1) Triangle A, 1, 2, 3, 1, 2-3 (a) superficial epigastric vessels; 1-2 (c) Poupart's ligament, lower half; 3-1 (d) obliterated hypogastric vessels. (2) Triangle B, 1, 6, 7, 1, 1-7 (b) Poupart's ligament; 1-6 (e) Cooper's ligament, lower portion; 6-7 (e) Cooper's ligament, upper portion and fascia and septum over iliapsoas muscle. (3) Triangle C, 5, 2, 1, 5, 2-1 (c) Poupart's ligament, lower half; 4-5 (d) obliterated hypogastric vessels; 5-2 (g) vas deferens.

anatomically and physiologically wrong. Anatomically, it closes only two of the weak spots. Though it occurs infrequently, most surgeons of considerable experience can recall cases in which an inguinal hernia had been repaired, the patient returned at a later date with a femoral hernia or vice versa. Physiologically, the surgeon knows that Poupart's ligament is considerably weakened, especially when fascial sutures are used by suturing into this structure. The fibers of this structure run longitudinally. Tension and sutures tear them apart. In the repair, if, the transversalis fascia is opened at the level of Poupart's ligament and the peritoneum held medially with a retractor, it is quite easy to drop down and pick up Cooper's ligament, suturing the conjoin tendon to this strong structure. (Fig. 3.) This may be likened to pulling the curtain clear to the base of the

window instead of only half way for an effective black-out. It is suggested that fascial or nonabsorbable sutures be used

FIG. 3. Repair of hernia with suture of conjoin tendon to Cooper's ligament (diagrammatic). (1) Poupart's ligament, upper portion; (2) external oblique fascia and muscle; (3) internal inguinal ring; (4) conjoined tendon sutured to Cooper's ligament; (5) sutures uniting the conjoin tendon to Cooper's ligament; (6) Cooper's ligament; (7) vas deferens.

here. The remainder of the operation may be completed according to the surgeon's particular fancy. It will be noted that the reflected portion of Poupart's ligament, at least in its proximal end, can be imbricated over part of the conjoin tendon and the external oblique imbricated over this, if so desired.

This procedure is applicable to all three types of hernias, eliminating the potential femoral hernia space also facilitating imbrication, and it furnishes a more secure anchorage for the key stitches.

CRYPTORCHIDISM

In the surgical treatment for the cure of the undescended testes, the difficult procedure is to get sufficient length to the cord to allow the testes to be placed in the scrotum without undue tension. A glance at triangle C in Figures 1 and 4 shows that the vas deferens traverses two

sides of the triangle i.e., 5, 6 and 1. The shortest distance is from 1 to 3 thereby actually giving from one and a half to two
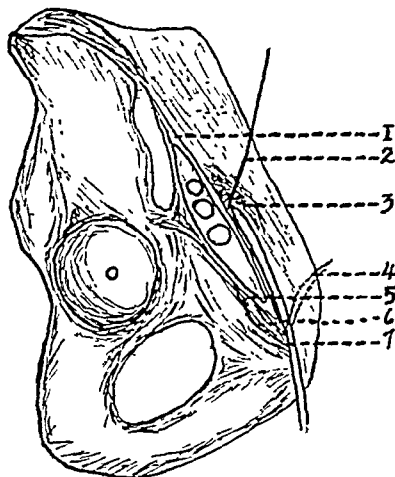
FIG. 4. Proposed "short cut" for course of vas deferens in orchidopexy: (1) Poupart's ligament; (2) superficial epigastric vessels; (3) internal inguinal ring; (4) vas deferens transplanted directly forward; (5) Cooper's ligament; (6) superior ramus of pubis; (7) vas deferens.

inches more mobility to the vas and the cord.

This may be accomplished by cutting and ligating the inferior epigastric vessels or better by passing the testes behind these vessels. In performing this procedure, if a hernia is present, it is advisable not to open the hernial sac, using this as a tractor, swinging the testes behind and medially, repairing the hernia as a last step before closure. The vas and cord usually get part of their blood supply from a branch of the inferior epigastric vessels. If possible this should be preserved but may be ligated if the spermatic artery has not been injured. The vas deferens can now be mobilized subperitoneally down to the origin at the base of the bladder. Great care should be exercised to preserve the blood vessels of the cord as ligation of the spermatic artery will result in atrophy of the testes. As noted in Figures 1 and 4 the vas deferens and cord now lie in a new route, i.e., a direct loop from the base of the bladder up over the medial end of

Poupart's ligament and down into the scrotum.

SUMMARY

A three triangle conception of the inguinal region is presented with the purpose of clarifying some of the vague and easily forgotten anatomical facts of this region. Based upon this conception and facts an appeal is made for the surgeon to do a complete herniorraphy at the time of the first operation rather than a partial one. Also an anatomically correct step is given which if used will aid the surgeon materially in percurring sufficient cord length in orchidopexy to enable him to place the testes in the scrotum without undue tension. The author claims no originality in these two procedures as they have been described and used for some time but it is his belief that their value is not generally understood nor made use of.

※⚜※

ONE of the characteristics of the vomiting of obstruction, as distinguished from other acute abdominal lesions, is that it tends to be more frequent and abundant. The rationale of this occurrence is readily apparent in the existence of a mechanical block in the bowel, which brings about a regurgitation of the stagnant intestinal content into the higher reaches of the alimentary canal. When accumulation occurs in the stomach, vomiting is copious usually.

From "Intestinal Obstructions" by Owen H. Wagensteen, Second Edition (Charles C. Thomas).

LEFT THORACO-ABDOMINAL STAB WOUNDS

Alex Steward, m.d.

On Thoracic Surgery Staff, Baroness Erlanger Hospital

CHATTANOOGA, TENNESSEE

THREE interesting and similar stab wounds of the left chest were admitted to the service of Erlanger Hospital in the year 1936.

CASE REPORTS

Case i. An obese, white, male, age twenty-six, was seen in the emergency room thirty minutes after being stabbed. He was pale but conscious and coherent in speech. There was a four inch knife slash from the right eye to the midcheek. The respirations were 28 per minute and the pulse rate, 124. The blood pressure was 94 systolic and 62 diastolic. The left side of the chest was immobile and there was a three-inch wound in the anterior axillary line, between the ninth and tenth ribs, plugged with omentum. The left side of the chest was hyperresonant and there was an absence of breath sounds. The right border of the heart was 1 cm. to the right of the sternum. The heart sounds were distant but regular. The patient was given morphine and coramine in the emergency room and sent to the operating corridor.

Under intravenous evipal anesthesia, the chest wound was extended into the left upper quadrant of the abdomen for about four inches. The cartilaginous connection between the ninth and tenth ribs had been severed. Exploration of the spleen showed a 0.5 cm. wound which did not completely penetrate the capsule on the anterosuperior surface. The other viscera were not disturbed. The herniated portion of the omentum, which extended through the diaphragm, was amputated. There was complete collapse of the left lung but no bleeding. The incision in the diaphragm was three inches long and was closed with mattress sutures of heavy linen. The intercostal and abdominal muscles were closed with chromic catgut and the skin with dermal sutures. Four hundred cc. of air was aspirated from the left pleural cavity after closing the chest wound and 300 cc. aspirated on the second, fourth and sixth postoperative days. On the twenty-second postoperative day the patient was dismissed to the police. Ten days later, an x-ray of the chest showed no pathological condition of the lung or diaphragm. The wound was firmly healed.

Case ii. A thirty-year old, male negro was seen in an emergency room about forty minutes after a friend cut him up. There were several superficial lacerations and from an immobile left chest a portion of omentum extended in the posterior axillary line between the ninth and tenth ribs. The left chest was hyperresonant and no breath sounds were audible. The heart was at the right side of the sternum and the sounds were weak but regular. The blood pressure was 90 systolic and 52 diastolic. Ten per cent glucose was given intravenously and coramine and caffeine intramuscularly. The patient was sent to the operating corridor.

Under intravenous evipal anesthesia, the extended omentum was slightly withdrawn, amputated, and held in the opening. Five and a half inch sections of the ninth and tenth ribs were removed, paralleling the wound and the pleural cavity opened. Blood clots were removed and blood aspirated. A three-inch laceration of the diaphragm was elongated for one inch, retracted and the left upper quadrant of the abdomen explored. The spleen had been completely perforated by the knife and was bleeding. There was no other visceral injury apparent. The spleen was removed through the diaphragm incision with perfect control of the vessels. The blood clots were removed from the upper quadrant and the diaphragm sutured. Two plain catgut stitches were inserted in a laceration of the left lower lobe of the lung. The chest wall was closed without drainage. Air and a moderate amount of serosanguineous fluid was aspirated intermittently from the chest until the patient was dismissed to the police on the nineteenth day and lost to hospital records.

It is interesting that the patient showed no blood changes other than a relatively high recuperative red cell count (3,680,000 to 4,520,000) during his hospitalization on a high caloric diet. Red blood cell increase and microscopic cell changes have been reported follow-

ing removal of traumatic spleens. There were no changes in this case.

CASE III. A twenty-two year old, male negro was seen in the emergency room with omentum protruding from a stab wound in the left anterior axillary line between the seventh and eighth ribs, about forty minutes after being stabbed. The left side of the chest was immobile, hyperresonant and breath sounds were absent except at the apex where they were diminished. The heart was displaced slightly to the right of the sternum but the sounds were of good quality, the rate 84 per minute, and the blood pressure 110 systolic and 80 diastolic. The left upper quadrant of the abdomen was tender and the muscles spastic. The patient was given morphine for pain and sent to the operating corridor.

Under ethylene anesthesia, the protruding omentum was cleansed, slightly withdrawn, clamped and the outer portion removed. Sections of the seventh and eighth ribs, approximately five inches long and paralleling the wound, were removed and the pleural cavity opened throughout the length of the incision. Pressure anesthesia had partially expanded the lung, but a two and one-half inch lacreation of the diaphragm was plugged with omentum. The protruding omentum was amputated and the abdomen explored by retracting the diaphragm wound. The stomach presented a one and one-half inch wound on its anterior aspect near the lesser curvature. The stomach was partially elevated into the pleural cavity and repaired with Lembert sutures. In the pleural cavity, some of the stomach's contents was expelled and this, together with blood clots, was washed out with normal saline solution. Blood clots were removed from the left upper quadrant. The diaphragm was closed with mattress sutures of chromic catgut. A small laceration in the lower lobe of the left lung was sutured and the chest wall closed. A soft rubber drain was inserted beneath the skin wound.

The postoperative convalescence was uneventful except for superficial wound infection and the patient was dismissed on the twenty-third postoperative day, after a negative x-ray examination and a completely healed wound.

The above emergency cases required immediate surgery. From examination, the weapons had penetrated into the abdomen

through the thorax. With a pneumothorax and cardiac embarrassment, supportive emergency room measures are usually necessary.

The type of anesthesia is important. A lung is collapsed. The patient is still partially shocked from sudden anoxemia and cardiac shift. Should intratracheal or any form of pressure anesthesia be used, re-expansion of the lung and perhaps bleeding may take place. On the other hand, a deoxygenated victim may need cardio-pulmonary support. In these cases of thoracotomys intravenous anesthesia and oxygen inhalation as indicated may be used. However, carefully controlled intratracheal anesthesia is preferable.

Surgical literature is prolific with reports of diaphragmatic hernias following wounds and injuries, (Christie,[1] Crook,[2] Harrington,[3] Hedblom,[4] Johnson[5]) but surprisingly little has been written on the immediate care of thoraco-abdominal wounds. Neugebaur[6] reports five cases (three gunshot and two stab wounds) which were operated upon within a few hours after their inception. The author used abdominal incisions and then extended the incision through the costochondral cartilage to explore the chest. Greene[7] reported six cases of stab wounds of the chest which extended through the diaphragm into the abdomen and were operated upon immediately. Two deaths occurred. In the first five cases, the writer explored the abdomen and then did a thoracotomy at the stab entrance. The sixth case was completed by the thoracic route alone.

The question of approach to a combined chest and abdominal wound is important. Lilienthal[8] sagely states that the site of the incision should depend on the location of the greatest amount of difficulty, either abdominal or thoracic, and that the individual case must be judged and no empirical rule can be followed. However, for a stab wound operated upon early, the direct thoracic approach offers some decided advantages for: (1) the lung is collapsed so that an adequate examination

of that organ can be made and the pleural cavity cleansed. (2) Exposure of the diaphragmatic wound is perfect. (3) Retraction of the diaphragmatic wound, and enlargement if necessary, brings immediately into view the viscus and abdominal area involved. Should there be indication for abdominal drainage, this may be done through small wounds in the abdominal wall directly to the involved site. (4) Early diaphragm repair avoids subsequent diaphragm operations which have a high mortality. (5) Chest wall closure is simple and, thanks to rapidly regenerating pericostal rib formation, a firm body wall maintained. Lung collapse is easily handled by aspiration as indicated.

CONCLUSIONS

1. In thoraco-abdominal stab wounds, immediate operation after shock is indicated.

2. Intravenous anesthesia and oxygen when necessary may be used although intratracheal is preferred.

3. Direct, transthoracic and transdiaphragmatic approach to the site of a thoraco-abdominal injury is advocated.

REFERENCES

1. CHRISTIE, G. W. Diaphragmatic hernia after bullet wound of chest. *Brit. M. J.,* 1: 599, 1925.
2. CROOK, J. L. Strangulated diaphragmatic hernia of traumatic origin; report of case. *Surg., Gynec. & Obst.,* 37: 185, 1923.
3. HARRINGTON, S. W., DORSEY, J. M. and SROHL, E. L. Left traumatic diaphragmatic hernia. *South. Clin. North America,* October, 1935.
4. HEDBLOM, C. A. Diaphragmatic hernia; a study of 378 cases in which operation was performed. *J. A. M. A.,* 85: 947, 1925.
5. JOHNSON, L. W. The post-traumatic abdomen; diaphragmatic hernia as a sequel of war injuries. *U. S. Nav. M. Bull.,* Vol. 34, October, 1936.
6. NEUGEBAUER, F. Uber Zwerchfellhernien und Zwerchfellverletzungen. *Beitr. z. klin. Chir.,* 144: 213–224, 1928.
7. GREEN, C. C. Stab wounds of the chest involving the diaphragm; with diaphragmatic hernia or evisceration. *J. A. M. A.,* 67: 178, 1916.
8. LILIENTHAL, H. Thoracic Surgery. Philadelphia, 1925. W. B. Saunders Co.

PRIMARY LYMPHOSARCOMA OF THE INTESTINE IN A BOY OF SEVEN

FOLLOW-UP OF NINE YEARS

HERMAN CHARACHE, M.D.

Associate Visiting Surgeon, Brooklyn Cancer Institute

BROOKLYN, NEW YORK

IN the January, 1935, issue of *The Americal Journal of Surgery*, a case of lymphosarcoma of the small intestine in a boy of seven was reported by the author. The rarity of the condition was discussed and 375 cases of lymphosarcoma of the intestine were found in the literature up to that time. Twenty-nine additional cases have been reported since, making a total of 404 cases.

CASE REPORT

The boy (F. P. No. 41916) was admitted to the Brooklyn Cancer Institute on December 4, 1933. Three weeks previous to admission to our Institution, he had been treated in the clinic of another hospital for a cold. During a routine examination a palpable tumor was found in the lower part of the abdomen. After a complete work-up, a gastrointestinal series revealed "some pathology connected with the small intestine, which did not appear to obstruct, but showed dilatation or distortion of the gut." An exploratory laparotomy performed by Dr. Dexter B. Davis revealed a freely movable tumor of the lower third of the ileum, measuring about three and one-half inches in diameter, and enlarged glands at the root of the mesentery. The tumor and about two inches of normal gut on either side were resected, followed by a lateral anastomosis. The pathological report of the tumor by Dr. James W. Denton was lymphosarcoma of the ileum. The diagnosis was later confirmed at our Institution.

A photograph of the patient, the resected segment of the intestine, and microphotograph of the tumor were included in the original article in 1935.

The patient received postoperative x-ray therapy at the Brooklyn Cancer Institute, where he has been followed in our clinic up to the present writing. Repeated roentgen examination of the lungs and gastrointestinal tract have failed to reveal any recurrence or metastasis. The last gastrointestinal series was done in February, 1942. The patient is now sixteen years old, well developed, attending school, and has no symptoms. He is still being carefully followed at the Brooklyn Cancer Institute.

Of the 404 cases of lymphosarcoma of the intestine reported in the literature, 103 had careful follow-ups. Of these, only fourteen survived for five years or more (13.6 per cent). Eight patients lived from seven to eleven years. The average duration of life was 16.7 months.

MARCH FRACTURE AS A COMPLICATION OF PREGNANCY

H. A. SWART, M.D.

In Charge of Orthopedic and Traumatic Surgery, Charleston General Hospital

CHARLESTON, WEST VIRGINIA

MARCH fracture, or March foot, was first described by Breithaupt,[1] a military surgeon, in 1855. He reported several cases of edematous and painful feet occurring in soldiers following long marches. He called it Fussgeschwulst and thought it was due to an inflammatory reaction of the tendon sheaths caused by trauma.

In subsequent years several other reports on this condition were made, but it was not until 1899 that Thiele[2] reported seventeen cases studied by the roentgen ray. Fifteen of these patients showed evidence of fracture of the metatarsals. There had been no history of trauma other than that produced by much walking while carrying heavy soldier's packs.

A detailed review of the literature will not be given as there have been several recent articles in which the previous reports have been thoroughly studied. Important among these is that of Meyerding and Pollock.[3]
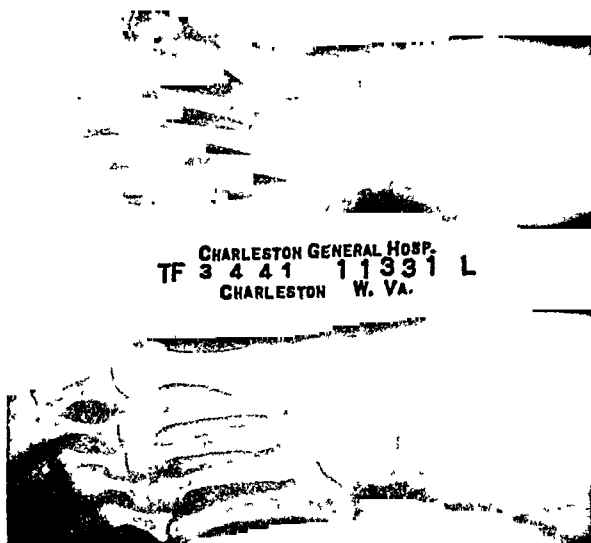
FIG. 1. Anteroposterior and oblique views of foot showing fracture of the distal half of the shaft of the second metatarsal with slight periosteal proliferation.

Various theories of etiology have been advanced. Zeitlin and Odessky[4] stated that the fracture resulted from overloading a foot weakened functionally and anatomically. Jansen's[5] theory was that spasm of the interosseus muscles led to hypertrophy of the muscle tissue and periosteum. Subperiosteal hemorrhages followed and this resulted in partial absorption of the bone, with resulting increase in brittleness and susceptibility to fracture.

Because of the fact that the true nature of March fracture has not been generally known in the past, serious mistakes have been made in diagnosis and treatment. Dodd[6] reported a case in which amputation was performed through a mistaken idea that sarcoma was present. Strauss[7] reported

a case in which a previous diagnosis had been made of sarcoma and amputation of the leg advised. There had been no history

weeks another roentgenogram will show definite evidence of callus formation at the site of fracture.
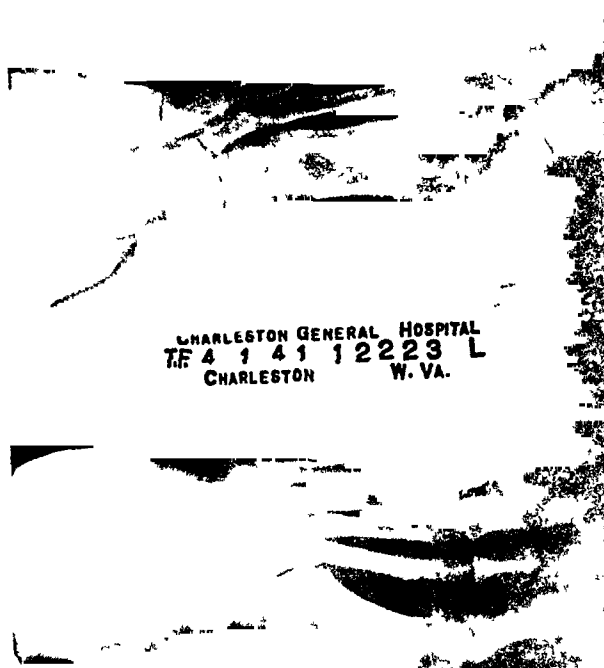
FIG. 2. Anteroposterior and oblique views of foot four weeks following application of cast, showing definite callus at the site of fracture of the shaft of the second metatarsal.

of trauma and no fracture line could be seen. Investigation for lues was negative, so the entire second metatarsal was removed. Other conditions which must be ruled out are chronic foot strain and tenosynovitis. In the former there is rarely any swelling and the latter seldom affects foot tendons. Roentgen ray evidence of fracture effectively rules out these two conditions and clinches the diagnosis.

The condition has been described in people of both sexes and in different walks of life. Pain in the foot follows much walking or weight bearing. There is accompanying swelling and tenderness. Roentgen rays may, or may not, show evidence of a fracture of the metatarsal shafts. The second metatarsal is usually involved, but all of the others may, at times, be implicated.

Many forms of treatment have been advocated, the most common being rest in a plaster cast. At the end of three or four

A reasonably complete review of the available literature in English reveals no case in which the condition was found in a pregnant woman. The case I wish to report is one of this type. Such cases must have occurred before as the necessary predisposing causes are found in practically all pregnant women. All of them gain weight and the feet of modern women are ill adapted to carry their normal weight, let alone the extra weight of a fetus. Obstetricians now advise their patients to walk more than they would ordinarily, and this brings in the factor of excessive weight bearing.

CASE REPORT

Mrs. T. C., a white woman, aged thirty, was first seen on March 4, 1941. She was seven months pregnant at that time and had been doing much walking to keep her weight down, on the advice of her physician.

For the previous eight days she had had swelling and pain in the left foot. This was confined to the dorsum of the foot and there had been no injury. It was gradually becoming worse. She had never had anything like it before. The swelling did not go down during the night and there had been no redness or other discoloration. No other joints were involved. On the evening of examination she complained of pain in the left sacroiliac region, for the first time. She had used infra red light and warm salts baths without relief.

Examination of the left foot showed diffuse swelling of the dorsum of the foot proximal to the toes. There was marked tenderness of the distal ends of the second and third metatarsals. There was no redness or increased local heat. Dorsiflexion of the toes was painful but plantar flexion did not cause pain. The blood vessels were easily palpated.

A roentgenogram taken the same day revealed a faint shadow along each side of the distal third of the shaft of the second metatarsal associated with a fine line running part way through the bone. These changes were barely visible on close scrutiny and can be shown only faintly in the reproduction of the roentgenogram. (Fig. 1.)

A diagnosis of March fracture was made and a short leg cast was applied from the ends of the toes to below the knee. A walking iron was placed on the cast and after the cast became hard she was instructed to walk on it. The sacroiliac pain was undoubtedly due to the strain of walking on a painful foot.

A few days later she called on the telephone saying that her foot was quite comfortable and that she had no pain. Thereafter, she got along very well and after wearing the cast four weeks it was removed. It was seen that both feet were slightly swollen, the left showing the greater degree of swelling. There was no tenderness over the fracture site, or elsewhere. Another roentgenogram showed very definitely the fracture of the shaft of the second metatarsal with abundant callus formation. (Fig. 2.)

She was advised to begin walking with a split shoe and crutches and to use warm baths, massage, and exercises to the foot at home. The foot quickly became normal and she was successfully delivered at term. Since then she has had no further trouble with her foot.

The use of the walking cast in this case represents, I believe, an improvement in treatment over those forms previously described. After the first few days this patient was quite comfortable and walked everywhere without any other support. Walking on the cast stimulates callus and keeps the joints from getting stiff. It also tends to prevent osteoporosis.

REFERENCES

1. BREITHAUPT. Zur Pathologie des Menschlichen Fusses. *Med. Ztg.*, 24: 169–175, 1855.
2. THIELE. Ueber Frakturen der Metatarsalknochen durch indirekte Gewalt (die Ursache der sogenannten Fussgeschwulst). *Deutsche med. Wchnschr.*, 15: 158–161, 1899.
3. MEYERDING, H. W. and POLLOCK, GEORGE A. March fracture, etc. *Surg., Gynec. & Obst.*, 67: 234–242, 1938.
4. ZEITLIN, A. and ODESSKY, I. Zur Differentialdiagnose Metatarsalfraktur oder morbus Deutschlaender. *Arch. f. Orthop.*, 34: 653–656, 1934.
5. JANSEN, MURK. March foot. *J. Bone & Joint Surg.*, 24: 262–227, 1926.
6. DODD, HAROLD. Pied force' or March foot. *Brit. J. Surg.*, 21: 131–144, 1933.
7. STRAUSS, F. H. Marching fractures of metatarsal bones, with a report of the pathology. *Surg., Gynec. & Obst.*, 54: 581–584, 1932.

AUTHOR INDEX TO VOLUME LIX

SUBJECT INDEX TO VOLUME LIX

(Bo. B.) = Bookshelf Browsing; (E.) = Editorial

The American Journal of Surgery

of Surgery

NEW SERIES, VOLUME LIX

JANUARY TO MARCH

1943

CONTENTS OF NEW SERIES, VOLUME LIX

ORIGINAL ARTICLES

iii

How 'Simple' Can
Solution-Preparation Be With Safety?

CUTTER SOLUTIONS IN *Saftiflasks*

An **UNBEATABLE COMBINATION** *for safety*
- A BIOLOGICAL LABORATORY'S SOLUTIONS —
THE SAFTIFLASK'S COMPLETE SIMPLICITY IN USE.

IS *solution-preparation just a case of mixing 'sugar' or salt with some distilled water, and then 'sterilizing'?*

Not if safety is your criterion!

Biological laboratory technicians know that intravenous solutions should be made and tested with the same exactitude as the most delicate vaccine or antitoxin. The large volume administered can, if only slightly contaminated, even with killed organisms, do more harm than might result from a contaminated vaccine or antitoxin of average dose.

Hence at Cutter Laboratories, one of America's oldest biological laboratories, each step in production, and the final product, are guarded by meticulous all-embracing tests . . . routine procedure in biological laboratories.

Cutter's specially designed Saftiflask — simplicity itself in use — is an additional safety guarantee. No gadgets to attach—no loose parts to wash, sterilize and assemble. Specify "in Cutter Saftiflasks."

CUTTER LABORATORIES • BERKELEY • CHICAGO • NEW YORK